PB86-232774

Development of Advisory Levels for Polychlorinated Biphenyls (PCBs) Cleanup

(U.S.) Environmental Protection Agency
Washington, DC

May 86

U.S. Department of Commerce
National Technical Information Service
NTIS

PB86-232774

EPA/600/6-86/002
May 1986

DEVELOPMENT OF ADVISORY LEVELS
FOR POLYCHLORINATED BIPHENYLS (PCBs) CLEANUP

Exposure Assessment Group
Office of Health and Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Washington, D.C.

REPRODUCED BY
NATIONAL TECHNICAL
INFORMATION SERVICE
U.S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA. 22161

TECHNICAL REPORT DATA

(Please read Instructions on the reverse before completing)

1. REPORT NO. EPA/600/6-86/002	2.	3. RECIPIENT'S ACCESSION NO. PB86 232774/AS
4. TITLE AND SUBTITLE Development of Advisory Levels for Polychlorinated Biphenyls (PCBs) Cleanup		5. REPORT DATE May 1986
		6. PERFORMING ORGANIZATION CODE EPA/600/21
7. AUTHOR(S) Seong T. Hwang, James W. Falco, Charles H. Nauman		8. PERFORMING ORGANIZATION REPORT NO.
9. PERFORMING ORGANIZATION NAME AND ADDRESS Office of Health and Environmental Assessment (RD-689) Exposure Assessment Group U.S. Environmental Protection Agency Washington, D.C. 20460		10. PROGRAM ELEMENT NO.
		11. CONTRACT/GRANT NO.
12. SPONSORING AGENCY NAME AND ADDRESS same as #9		13. TYPE OF REPORT AND PERIOD COVERED
		14. SPONSORING AGENCY CODE EPA/600/21

15. SUPPLEMENTARY NOTES

16. ABSTRACT

This document presents background information used in developing advisory levels of PCBs in soil estimated to be permissible in protecting public health. The results of exposure assessment and health effects studies are combined to arrive at the permissible levels of PCBs. Health effects studies conducted using animals for the duration of 10-30 days are used to determine the 10-day advisory levels for PCB cleanup. The long-term advisory levels are based on the carcinogenic risk evaluations.

Exposure pathways considered in estimating the 10-day and long-term average daily intakes include soil ingestion, inhalation, dermal contact, ingestion of contaminated food, and ingestion of water. Exposure to drinking water contaminants is presumed to occur independently of other pathways, because water could come from a clean public water system. The exposure pathways most pertinent to the evaluation of permissible PCB levels in soil are soil ingestion, vapor inhalation, and contaminant contact with human skin.

The currently available modeling techniques considered most appropriate within the constraints of availability of input data are used to estimate exposures. PCB advisory levels are presented as ranges of values to reflect the difference in soil-air partition coefficients depending on soil type, different types of commercial Aroclors, and variations in the soil ingestion rate.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group

18. DISTRIBUTION STATEMENT Release to the public	19. SECURITY CLASS *(This Report)* Unclassified	21. NO. OF PAGES 216
	20. SECURITY CLASS *(This page)* Unclassified	22. PRICE

EPA Form 2220-1 (9-73)

DISCLAIMER

This document has been reviewed in accordance with the U.S. Environmental Protection Agency's peer and administrative review policies and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

CONTENTS

Tables v

Foreword vii

Preface. ix

Abstract x

Authors and Reviewers. xi

1. Executive Summary. 1-1

2. Introduction 2-1

3. Chemical Compositions. 3-1

4. Production 4-1

5. Uses 5-1

6. Disposal 6-1

7. Chemical and Physical Properties 7-1

8. Environmental Distribution 8-1

9. Environmental Fate and Transport 9-1

10. Toxicology 10-1

11. Existing Standards and Guidelines 11-1

12. Exposure Assessment Methodology. 12-1

  12.1 Estimation of Exposures for Contaminated Sites 12-3
  12.2 Determination of Permissible Pollutant Level in Soil . . 12-10
  12.3 Incorporation of Time-Varying Parameters 12-11
  12.4 PCB Advisory Evaluations 12-14

13. Water Quality Limits 13-1

14. Leachate Contamination of Groundwater. 14-1

15. Soil Ingestion Pathway 15-1

CONTENTS (continued)

16. Inhalation Pathway 16-1

16.1. Intake by Air Exposure Route. 16-1
16.2. Emission Evaluation Scenarios 16-2
16.3. Air Dispersion Modeling 16-8
16.4. Air Exposure Evaluation 16-10

17. Dermal Contact Pathway 17-1

18. Comparison of Exposures by Soil Ingestion, Inhalation, and Dermal Contact 18-1

19. Results. 19-1

19.1 Derivation of Permissible Soil Contamination 19-1
19.2 Summary of Results 19-4

20. Limitations of Application 20-1

21. References. 21-1

APPENDICES

A. Models Used in Air Release Rate Calculations A-1

B. Example Emission Rate Calculations for Four Studied Scenarios B-1

C. Summary of Computer Runs for Each Aroclor and at Each Value of Soil-Air Partition Coefficients C-1

D. Health Advisories for PCBs in Soil (Prepared by M.L. Dourson, Environmental Criteria and Assessment Office, Cincinnati, OH) D-1

TABLES

1. Permissible PCB soil contamination levels (uncovered surface contamination). 1-6

2. Permissible PCB soil contamination levels (25-cm-thick clean cover). 1-7

3. Approximate composition of Aroclors 3-3

4. Chemical and physical properties of PCBs. 7-3

5. Solubility of chlorobiphenyls in water. 7-6

6. PCB monitoring in ambient air by NYSDEC 9-2

7. Existing PCB standards and guidelines 11-3

8. Maximum lifetime risk for ingesting soil contamination at different PCB levels 15-1

9. Maximum daily PCB intake by ingestion of soil at various PCB concentrations 15-2

10. Comparison of PCB intakes by ingestion and inhalation routes for acute effects. 16-3

11. Comparison of PCB intakes by ingestion and inhalation routes for carcinogenic effects 16-4

12. Concentration of PCBs in soil at saturation vapor pressure based on K_d = 1,000 cm^3/g. 16-7

13. PCB emission rates from 1 μg/g PCB soil at different control levels. 16-7

14. Values of constants for standard deviation expression as a function of downwind distance and stability condition 16-10

15. Ambient PCB concentrations at different locations (PCB in soil = 1 μg/g, PCB-1254). 16-11

16. Comparison of intakes by various exposure routes. 18-1

17. Evaluation conditions for each Aroclor. 19-5

18. Low and high values of air-soil partition coefficient used in the evaluation 19-6

19. Permissible PCB-1242 soil contamination levels (uncovered surface contamination). 19-7

20. Permissible PCB-1248 soil contamination levels (uncovered surface contamination). 19-8

21. Permissible PCB-1254 soil contamination levels (uncovered surface contamination). 19-9

22. Permissible PCB-1260 soil contamination levels (uncovered surface contamination). 19-10

23. Permissible PCB-1242 soil contamination levels (25-cm-thick clean soil cover) 19-11

24. Permissible PCB-1248 soil contamination levels (25-cm-thick clean soil cover) 19-12

25. Permissible PCB-1254 soil contamination levels (25-cm-thick clean soil cover) 19-13

26. Permissible PCB-1260 soil contamination levels (25-cm-thick clean soil cover) 19-14

FOREWORD

The Exposure Assessment Group (EAG) of EPA's Office of Research and Development has three main functions: 1) to conduct exposure assessments; 2) to review assessments and related documents; and 3) to develop guidelines for Agency exposure assessments. The activities under each of these functions are supported by and respond to the needs of the various EPA program offices. In relation to the third function, EAG sponsors projects aimed at developing or refining techniques used in exposure assessments, and at applying these techniques to develop health-based advisory levels for contaminant cleanup. This document is one of these projects and was done for the Office of Solid Waste and Emergency Response.

Polychlorinated biphenyls (PCBs), commercially known as Aroclors, consist of mixtures of chlorinated biphenyl compounds. Many sites contaminated by PCBs remain contaminated because of PCB persistence in the environment. Although commercial PCB production has been banned by the Toxic Substances Control Act, continued use in previously existing commercial equipment can result in spills which require cleanup. EPA has become increasingly involved in the discovery, assessment, and cleanup of these sites.

The purpose of this document is to provide advisory levels for PCB cleanup, and to describe the detailed technical and scientific rationale and methods used in developing these advisory levels for PCBs in contaminated soil. This project required development of exposure and risk assesment methodology related to hazardous waste and spill sites, and analyses of health effects data. The advisory levels and the assessment methodology thus developed will help EPA set priorities in PCB spill and cleanup management, and address other PCB contaminant problems.

Michael Callahan, Director
Exposure Assessment Group

PREFACE

The Exposure Assessment Group of the Office of Health and Environmental Assessment (OHEA) has prepared this development document for advisory levels for polychlorinated biphenyls (PCBs) cleanup at the request of the Office of Emergency and Remedial Response. This document summarizes the procedures concerning multimedia exposure assessments for PCB-contaminated sites, and literature information on chemical and physical properties and health effects pertinent to evaluation of exposures to PCBs. The purpose of this document is to serve as a technical and scientific basis for developing health-based advisory levels for PCBs in soil at spill or cleanup sites. The literature search supporting this document is current to May 1986.

ABSTRACT

This document presents background information used in developing advisory levels of PCBs in soil estimated to be permissible in protecting public health. The results of exposure assessment and health effects studies are combined to arrive at the permissible levels of PCBs. Health effects studies conducted using animals for the duration of 10-30 days are used to determine the 10-day advisory levels for PCB cleanup. The long-term advisory levels are based on the carcinogenic risk evaluations.

Exposure pathways considered in estimating the 10-day and long-term average daily intakes include soil ingestion, inhalation, dermal contact, ingestion of contaminated food, and ingestion of water. Exposure to drinking water contaminants is presumed to occur independently of other pathways, because water could come from a clean public water system. The exposure pathways most pertinent to the evaluation of permissible PCB levels in soil are soil ingestion, vapor inhalation, and contaminant contact with human skin.

The currently available modeling techniques considered most appropriate within the constraints of availability of input data are used to estimate exposures. PCBs advisory levels are presented as ranges of values to reflect the difference in soil-air partition coefficients depending on soil type, different types of commercial Aroclors, and variations in the soil ingestion rate.

AUTHORS AND REVIEWERS

The Exposure Assessment Group of the Office of Health and Environmental Assessment was responsible for preparing this document.

AUTHORS

Seong T. Hwang
Exposure Assessment Group
Office of Health and Environmental Assessment
U.S. Environmental Protection Agency

James W. Falco
Office of Environmental Processes and Effects Research
U.S. Environmental Protection Agency

Charles H. Nauman
Exposure Assessment Group
Office of Health and Environmental Assessment
U.S. Environmental Protection Agency

REVIEWERS

The following individuals provided review comments and criticisms during several peer-review processes to which this document was subjected.

Don R. Clay, Director
Office of Toxic Substances
U.S. Environmental Protection Agency

Joseph A. Cotruvo, Director
Criteria and Standards Division
Office of Drinking Water
U.S. Environmental Protection Agency

Yoram Cohen
Department of Chemical Engineering
University of California, Los Angeles
Los Angeles, California

Barbara Davis
Office of Waste Programs Enforcement
U.S. Environmental Protection Agency

Karen Hammerstrom
Exposure Evaluation Division
Office of Toxic Substances
U.S. Environmental Protection Agency

Krishan Khana
Health Effects Branch
Office of Drinking Water
U.S. Environmental Protection Agency

Russell Kinerson
Office of Toxic Substances
U.S. Environmental Protection Agency

William Marcus
Office of Drinking Water
U.S. Environmental Protection Agency

Robert E. McGaughy
Carcinogen Assessment Group
Office of Health and Environmental Assessment
U.S. Environmental Protection Agency

Mary Lund Mortensen
Agency for Toxic Substances and Disease Registry
Atlanta, Georgia

Debdas Mukerjee
Environmental Criteria and Assessment Office--Cincinnati
U.S. Environmental Protection Agency

Judith A. Nelson, Director
Regulatory Coordination Team
Office of Pesticides and Toxic Substances
U.S. Environmental Protection Agency

Arnett Nold
Office of Toxic Substances
U.S. Environmental Protection Agency

Edward V. Ohanian
Health Effects Branch
Office of Drinking Water
U.S. Environmental Protection Agency

Suresh Rao
Soils Department
University of Florida
Gainesville, Florida

Danny Reible
Department of Chemical Engineering
Louisiana State University
Baton Rouge, Louisiana

Charles Ris
Carcinogen Assessment Group
Office of Health and Environmental Assessment
U.S. Environmental Protection Agency

Jerry F. Stara
Environmental Criteria and Assessment Office--Cincinnati
Office of Health and Environmental Assessment
U.S. Environmental Protection Agency

Louis Thibodeaux
Hazardous Waste Research Center
Louisiana State University
Baton Rouge, Louisiana

Edwin F. Tinsworth, Deputy Director
Office of Toxic Substances
U.S. Environmental Protection Agency

1. EXECUTIVE SUMMARY

This report has been prepared in response to a memorandum dated April 9, 1985, from the Office of Emergency and Remedial Response (OERR), requesting that the Office of Health and Environmental Assessment (OHEA) develop advisory levels for polychlorinated biphenyls (PCBs) which can be used as guidelines for initiating removal action for sites contaminated with PCBs. Interested offices within EPA, including OERR, have advised OHEA that these advisory levels for PCBs cleanup should be developed based on considerations of public health protection from short-term and long-term exposures. The advisories presented in this report include permissible levels of PCBs in soil corresponding to 10-day and lifetime acceptable intakes.

Exposure routes considered in developing these advisory levels include drinking water, ingestion of PCB-contaminated soil by children and adults, and inhalation of ambient air contaminated with PCBs. Other exposure routes, such as dermal exposure, food intake, and ingestion of fish which have bioaccumulated PCBs, are considered in relation to their importance and their relevance to the present document. In view of the high bioaccumulation factor for PCBs, the consideration of bioaccumulation is important in setting PCB levels in surface water in which aquatic animals live. If one of these routes is a controlling factor in relation to the exposure route or human intake considered, the advisories need to be reevaluated.

Commercial-grade PCBs marketed as Aroclors in the United States are mixtures of many chlorinated biphenyl compounds in various proportions. Each PCB compound may exhibit its own toxicological characteristics and physical and chemical properties. This fact complicates the exposure analysis in deriving the allowable concentrations in drinking water and soil. It is conceivable that chemical and physical properties reported in the literature for each Aroclor designation represent an average property for the mixture. To define the variability of safe levels for contamination by different Aroclor designations, exposure analyses have been performed for several Aroclors: Aroclor 1242, 1248, 1254, and 1260. The steps used in developing the advisories include: (1) the evaluation of toxicological effect studies, (2) exposure analysis for PCB intake from drinking water, soil ingestion, air inhalation, and dermal contact, and (3) risk assessment combining the toxicology studies and exposure analysis.

Ten-day noncancer health advisories are based on the short-term acceptable intake (AI) derived from studies of animals treated with Aroclor-1254 for no more than 30 days to examine noncarcinogenic effects. This AI value, which forms the basis for establishing permissible levels of PCBs in soil, is 0.1 and 0.7 mg/day for a 10-kg child and a 70-kg adult, respectively. The permissible PCB concentrations for each carcinogenic risk level are based on the potency factor of 4 $(mg/kg\cdot day)^{-1}$ rounded off from two independent evaluations based on an Aroclor-1260 study.

It is likely that not all of the PCBs ingested or inhaled by humans are absorbed. Proper calculations of absorption rate and hence exposure should be based on realistic pharmacokinetics-type models to determine intake. Lack of experimental data with which to estimate the parameters needed in the pharmacokinetics models has prevented their applications to the analysis for PCB absorptions through human exchange boundaries. Future work should consider these models. Although most animal studies (in rats and mice) on the extent of absorption in the gastrointestinal tract show absorption in excess of 90%, there are two experiments on monkeys reporting less than 88% absorption in one case and less than 13% and 40% absorption for a specific congener in another case, based on the analysis of feces and urine. Vehicles used in administering

PCBs were not specified. It is likely that the high adsorption characteristics of PCBs on soil could retard the absorption rate in the human intestinal tract. In the risk analysis performed in the present study, the absorption rate for humans after ingestion of PCB-contaminated soil is considered to be 30%.

Absorption from dermal exposure has been reported to be as significant as from other routes of exposure, but little information is available for the quantitative evaluation of dermal absorption rates. Five percent dermal absorption is assumed for soil contaminants in contact with human skin. The dermal absorption rate of contaminants present on soil is presumed to be less than that for contaminants spilled on skin in pure form. Inhalation studies using PCB aerosols show that the absorption of PCBs from inhalation exposure readily occurs. In the present analysis, an absorption factor of 50% is assumed for absorption of PCB vapors after inhalation into human lungs.

The circumstances under which human exposure occurs are divided into three classes depending on population distribution: (1) Exposure occurs on-site. This can be further subdivided into: (a) sites which are readily accessible to children, and, hence, the soil from which will be subject to ingestion, dermal contact, and inhalation, and (b) sites for which there is no possibility of soil ingestion, and, hence, exposure is only through inhalation; (2) sites which no population is assumed to enter within a radius of 0.1 km from the site; and (3) sites which no population is assumed to enter within a radius of 1 km from the site.

The soil ingestion rates used for Class (1)(a) evaluations are 3 and 0.6 g/day. The former is a value based on data from a study of an adult person with pica, while the latter represents a long-term average value for soil ingestion. If sites are not accessible to populations at distances of 0.1 km or 1 km from the site, as in Classes (2) and (3) above, it is assumed that no ingestion of contaminated soil occurs and the exposure route is that of inhalation.

The emission rate of volatilized PCBs can be considerably reduced by covering the contaminated soil by low-porosity uncontaminated soil or clay material. The reduction in the emission rate will result in a decrease in ambient air concentrations of PCBs by the action of blowing winds. When PCB-contaminated material is directly exposed to the atmosphere, the PCB levels in soil required to maintain the same level of exposure will be less than those expected when the PCB-contaminated material is covered with low-permeability material of appropriate thickness. The cover would also serve as a deterrent to soil ingestion and direct dermal contact.

The depletion of PCBs from soil caused by volatilization is accounted for in the exposure analysis by solving a partial differential equation simulating PCB vapor diffusion through the soil air-phase pores, and the distribution of PCBs between air and soil phases. Boundary conditions assume that the air-phase resistance is relatively small compared to the diffusional resistance in the soil air-phase pores. The available experimental data reasonably follow the time-emission rate relationship predicted from the models based on this assumption. Since the depletion rate varies over time, it is averaged over the exposure period. Depletion averaged over a period of time should lead to a lesser inhalation exposure than that based on the model assuming that depletion does not occur.

The worst-case emissions would occur when the contaminated soil is initially exposed to the atmosphere and the soil is contaminated up to the conditions exhibiting saturation vapor pressure. A constant emission rate can be assumed if the vapor-phase concentration maintains a constant value at the surface of soil contamination for time-varying emission rates. Calculations corresponding to Classes (1), (2), and (3) for exposure possibilities with surface contamination are repeated at an assumed 25-cm thickness of a soil cover initially free from PCB contamination. Among many factors affecting the emission rate (including vapor pressure, soil-air partition coefficient, Henry's Law constant, etc.), the value of the soil-air partition coefficient shows the most wide-ranging variation, because of the variation of the experimental soil-water partition coefficient available in the literature for soil textures ranging from 40 to 1,000 cm^3/g.

The method for determining the permissible PCB levels in soil, which combines the routes of soil ingestion, inhalation, and dermal exposure, has been computerized to avoid the necessity for hand calculations.

The results of these computer calculations are summarized in Tables 1 and 2, which have been prepared using different combinations of the following variables:

(1) Surface contamination representing a situation where the contaminated soil surface has been left uncovered after removal action.

(2) 25-cm (10-inch) clean cover applied, representing a situation in which clean soil material is used on top of the contaminated soil surface.

(3) Two different soil ingestion rates (3 and 0.6 g/day) for Class (1)(a), corresponding to sites accessible to children.

(4) Different AI levels (short-term AI, and AIs at different cancer risk levels).

(5) Four Aroclors (Aroclor 1242, 1248, 1254, and 1260).

(6) Two selected values of the soil-air partition coefficient, representing the high and low values.

TABLE 1. PERMISSIBLE PCB SOIL CONTAMINATION LEVELS (UNCOVERED SURFACE CONTAMINATION)

Location and route of human exposure	Permissible levels (µg PCB/g soil) corresponding to					
	Noncancer short-term[a] acceptable intake (µg/day)[b]		Cancer risk specific doses (µg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	25-100[f]	510-730	0.008-0.01	.08-0.1	0.8-2	8-17
- Soil ingestion[d], inhalation[e]	42-420	2100-3000	0.01-0.06	0.1-0.6	1-6	35-61
- Inhalation only[e]	47-vs[g]	vs	0.01-0.2	0.1-2.0	1-20	77-470
0.1 km from contaminated site - Inhalation only[e]	vs	vs	2.0-220	90-2.2×10^4	7.7×10^3-vs	8.7×10^5-vs
1 km from contaminated site - Inhalation only[e]	vs[g]	vs	220-1.3×10^3	2.2×10^4-1.3×10^5	vs	vs

[a] Short-term ≅ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days' exposure per year.

[f] Ranges result in each case because 1) four PCBs (1242, 1248, 1254, 1260) are considered, each with a different vapor pressure, and 2) high and low values for soil-air partition coefficient are used in the calculations.

[g] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquids for the limit.

TABLE 2. PERMISSIBLE PCB SOIL CONTAMINATION LEVELS
(25-cm-THICK CLEAN COVER)

Location and route of human exposure	Permissible levels (µg PCB/g soil) corresponding to					
	Noncancer short-term[a] acceptable intake (µg/day)[b]		Cancer risk specific doses (µg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	110-200[f]	800-1400	0.01-0.2	0.1-2.0	1-17	22-vs
- Soil ingestion[d], - inhalation[e]	450-vs[g]	3100-vs	0.02-0.6	0.2-6.0	1.0-48	93-vs
- Inhalation only[e]	vs	vs	0.02-1.0	0.2-vs	2.0-vs	770-vs
0.1 km from contaminated site - Inhalation only[e]	vs	vs	1-vs	620-vs	vs	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	vs	vs	vs	vs

[a] Short-term ≅ 10-day intake.
[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.
[c] Children ages 1-5, with pica (consuming 3 g soil/day).
[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).
[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days' exposure per year.
[f] Ranges result in each case because 1) four PCBs (1242, 1248, 1254, 1260) are considered, each with a different vapor pressure, and 2) high and low values for soil-air partition coefficient are used in the calculations.
[g] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquids for the limit.

(7) Exposures for 10 days after cleanup or spill of contaminants for short-term advisories.

Table 1 shows the range of values for permissible PCB concentrations in soil when the soil is contaminated up to the surface in contact with the atmosphere and is left uncovered. Table 2 represents the case where the contaminated soil left at the site, or after remediation, is covered with a 25-cm (10-inch) clean soil layer. The ranges in both tables result from the use of four Aroclors and the use of high and low values for the soil-air partition coefficient. Other factors reflected in the ranges are differences in vapor pressures and Henry's Law constants for each Aroclor. The permissible PCBs levels in soil specific to each combination of the scenarios are compiled in Appendix C, as obtained from computer simulations.

The symbol "vs" in Tables 1 and 2 indicates that no upper-bound limit for PCB concentrations in soil can be derived from the exposure evaluation, because the PCB concentration in soil is above the vapor saturation concentration. There are two reasons for such a result. First, the emission rate cannot exceed the upper-bound value which can be expected when the air-phase concentration of PCBs at the contaminated soil surface is maintained at the vapor saturation point. The concentration at the vapor saturation point corresponds to the vapor pressure concentration. Second, when the cover is applied, not only is the emission rate retarded, but also the concentration of PCBs in soil being ingested is controlled by the amount of PCBs adsorbed on soil in equilibrium with the air phase being emitted. Therefore, the concentration of PCBs in the initially clean soil material cannot exceed the concentration in equilibrium with saturated vapor.

In actuality, the "no upper limit," or the level above vapor saturation, designated by vs, should be interpreted with great care. The assumptions used in the exposure evaluation are critical. They include but are not limited to: (1) no soaking of clean cover by liquid PCBs for the thickness of 25 cm; (2) no disturbance of cover material by construction activities or children digging the ground; (3) no exposure to initial spills when 25 cm of clean cover (Table 2) is assumed; (4) no population enters the area within the respective radius of distances from the site; and (5) the cover material is at least equivalent to soil material.

From a practical point of view, Assumption 1 is tantamount to requiring the presence of no free liquids in the soil, which may otherwise result in the phenomenon of "wicking." Since the ranges shown in Tables 1 and 2 are dependent upon the type of Aroclors and the values of the soil-air partition coefficient, site-specific or Aroclor-specific information should be used to establish an appropriate level of PCBs for that particular condition. The methodology for performing site-specific exposure evaluations is presented. Computer outputs for the selected Aroclors under the ranges and conditions of common environmental concern are presented in Appendix C, and can easily be used to find the permissible concentrations in soil suitable to particular situations.

Table 1, for example, can be interpreted as follows:

(1) When the site is amenable to access by children with possibilities of ingesting the contaminated soil exposed to the atmosphere, and when exposure occuring to the children by inhalation and dermal contact is accounted for, the permissible PCB levels in soil should range from 25 to 100 μg/g and 42 to 420 μg/g for prevention of noncancer effects from 10-day exposure for a child with an average weight of 10 kg ingesting soil at the rates of 3 and 0.6 g/day, respectively. For cancer effects, permissible levels in soil for a lifetime exposure to PCBs resulting from ingestion of and dermal contact with contaminated soil and inhalation of contaminated air should range from 0.08 to 0.1 μg/g

and 0.1 to 0.6 µg/g, corresponding to an upper-bound risk estimate of 10^{-6} at assumed soil ingestion rates of 3 and 0.6 g/day, respectively. The specific level will depend on the types of Aroclor present, the likely ingestion rate, and the extent of soil-air partitioning. For sites in which there is no possibility of soil ingestion, PCB levels in soil, based on the inhalation route only, should range from 47 µg/g to no limit value for a 10-day exposure for a child with an average weight of 10 kg, and correspond to no limit value for an adult with an average weight of 70 kg. The permissible levels of PCBs in soil, based on the inhalation pathway only, range from 0.1 to 2 µg/g, corresponding to a lifetime AI at a risk factor of 10^{-6}. Again, the level will be dictated by the types of Aroclor present and the specific characteristics of the site involved.

(2) If there is no possibility of a population entering the contaminated site within a radius of 0.1 km from the site, the PCB levels in soil can remain at no limit value and 90 to 2.2×10^4 µg/g, without exceeding 10-day AI and lifetime AI at 10^{-6} risk, respectively.

Similar interpretations can be made for the results applicable to sites without affected population up to 1 km from the site, and to the carcinogenic risks listed at 10^{-4}, 10^{-5} and 10^{-7}.

The short-term AI levels (100 µg/g day for a child and 700 µg/g day for an adult) used in this report to develop 10-day advisories based on noncancer effects are derived from animal studies, which collectively indicate that the experimental threshold for adverse effects of Aroclor 1254 is at or near a dose of 1.0 µg/kg body weight. Using this dose as a No Observed Adverse Effect Level (NOAEL) and a safety factor of 100, the 10-day AI levels for noncancer effects described above (100 and 700 µg/day) were computed and serve

Advisory levels for 1-day and lifetime noncancer effects cannot be derived at this time because of the insufficiency of the available data. However, in view of the experimental duration, the 10-day advisories may well be used for the 1-day advisories.

2. INTRODUCTION

The purpose of this document is fourfold: (1) to provide background information compiled in the process of developing permissible health advisories for polychlorinated biphenyls (PCBs) in soil and drinking water, in response to a request from the Office of Emergency and Remedial Response, (2) to outline the procedures used in developing the advisories, (3) to list pertinent input data necessary in carrying out the exposure analyses and in setting the allowable concentration limits, and (4) to present an outline summary of the results obtained from computer simulations of the techniques used.

The information and methods presented are intended for use in setting safe advisory levels to protect public health from short-term, longer-term, and lifetime exposures to PCBs released from hazardous waste facilities or from spills at previously contaminated sites. Particular interests pertain to levels of PCBs allowable in contaminated soil, and the potential of PCB migration to groundwater from PCB-contaminated or hazardous waste facilities. These advisories are not concerned with setting PCB limitations in sediments contaminating surface water, which can be a source of bioaccumulation of PCBs in aquatic animals.

The analyses presented in this report provide the basis for deriving PCB levels allowable in soil and drinking water, which are likely to be primary sources of exposure pathways. PCB problems that may exist in rivers and estuaries because of contaminated sediments are not dealt with in these analyses. The analyses mainly address health concerns at hazardous waste sites or at sites with contaminated soil. The primary health impacts considered include adverse impacts associated with ingestion of contaminated soil, inhalation of ambient air, and dermal contact with the soil. Other exposure routes, such as drinking water, food intake, and ingestion of bioaccumulated fish are considered to the extent that the pathways are relevant. PCBs that have migrated from contaminated sites to various exposure media are evaluated for short-term and lifetime impacts to arrive at corresponding advisory level values.

The total dose of PCBs is obtained by summing each dose from all major exposure pathways, and is compared with acceptable intakes (AI) judged from the health effects information available in the literature. A longer-term AI considered most appropriate in deriving 10-day noncarcinogenic advisories is used in the exposure evaluation. Advisories for protecting against carcinogenic risks are similarly derived at various risk levels based on the potency factor.

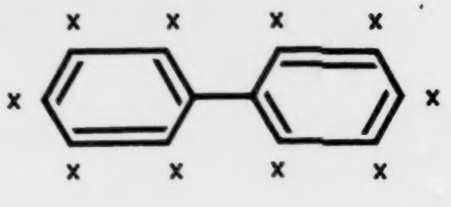
This report is not intended to address the achievability of the safe levels developed. Although the report contains a brief statement, taken from the available literature on analytical capability, control technology, and environmental distribution of PCBs, the data base seems insufficient to make a generalization concerning the level of PCB cleanup achievable in practice.

The Exposure Assessment Group distributed three earlier drafts of this document under the title of "Development of Health Advisories for Polychlorinated Biphenyls (PCBs)" for internal review and comment on May 9, 1985 and July 25, 1985, and under the title of "Development of Health Advisories for Polychlorinated Biphenyls (PCBs) Cleanup" on December 16, 1985. As a result of the comments from the Office of Drinking Water, on the December 16, 1985 draft, the 1-day advisories have been replaced by 10-day advisories because no data could be found indicating that 3,4,5,3',4',5'-hexachlorobiphenyl, used for development of the 1-day advisories, is a component in commerical Aroclors. This final draft reflects changes made to incorporate comments from the Office of Toxic Substances, the Office of Emergency and Remedial Response, and OHEA's

Environmental Criteria and Assessment Office.

3. CHEMICAL COMPOSITIONS

A polychlorinated biphenyl (PCB) is any member in a family of organic compounds with two or more chlorine substitutions on biphenyl rings, and can be typified by the following chemical structure:



The symbol, x, in the structural formula represents possible positions of chlorine that can be substituted for hydrogen, which is one of the basic elements of aromatic hydrocarbons. Based on the possible distribution of substituted chlorine atoms on two benzene rings of biphenyl, it is calculated that there could theoretically be 209 types (congeners) of PCBs.

Patents disclose that PCBs are prepared by the chlorination of biphenyl in the presence of a catalyst. The process yields a complex mixture of chlorinated biphenyl compounds. It is unlikely, however, that all combinations of chlorinated biphenyls would be formed in the chlorination process. Although the crude mixture is purified to remove reaction impurities, the resulting product is still a mixture of chlorinated biphenyls in various proportions. Their compositions depend upon the chlorination conditions.

Commercial-grade PCBs, consisting of mixtures of different composition, are sold under the trade name Aroclors. Impurities such as chlorinated dibenzofurans and chlorinated naphthalenes are known to exist in commercial PCBs. The sole producer of Aroclors in the United States for the period 1957 to 1972 was the Monsanto Chemical Company. Their products are characterized by four-digit numbers. The first two numbers represent the type of molecule (12 = biphenyl-based; 54 = terphenyl-based; 25,44 = blends of PCBs and chlorinated terphenyls); and the last two digits refer to the percentage of chlorine by weight. PCB products are also manufactured in other countries, including Germany, France, Japan, and the U.S.S.R.

Table 3 illustrates approximate compositions of individual biphenyls for some Aroclors (U.S. EPA, 1976b). Although one might expect some 140 to 150 separate congeners in an Aroclor, the actual analysis of Aroclor 1248, for example, identified less than 50 peaks in the high-resolution gas chromatograph using a typical Aroclor 1248 sample (U.S. EPA, 1976b). No compounds which can be formed by addition of chlorine rather than substitution were found in a detailed study of PCBs (U.S. EPA, 1976b). It is suspected that the conditions prevailing during industrial manufacturing of PCBs do not favor the formation of addition compounds, or that these latter compounds might have been destroyed in the step used to purify the Aroclor. In constrast to the analysis shown in Table 3, another publication reports an analysis of Aroclor 1221 to contain 12.7% biphenyl, 47.1% monochlorophenyls, and 40.2% dichlorophenyls (Hutzinger et al., 1974).

Major PCB components in foreign products bearing the names of Kanechlor and Phenoclor for Japanese and French products, respectively, have been identified. The number of the major components separated from Kanechlor 400 is five, and that from Phenoclor DP6 is seven.

TABLE 3. APPROXIMATE COMPOSITION OF AROCLORS

	Percent by weight for Aroclor designation					
Chlorobiphenyl	1221	1242	1248	1254	1260	1016
$C_{12}H_{10}$	11	<0.1		<0.1		<0.1
$C_{12}H_9Cl$	51	1		<0.1		1
$C_{12}H_8Cl_2$	32	16	2	0.5		20
$C_{12}H_7Cl_3$	4	49	18	1		57
$C_{12}H_6Cl_4$	2	25	40	21		21
$C_{12}H_5Cl_5$	0.5	8	36	48	12	1
$C_{12}H_4Cl_6$	ND	1	4	23	38	<0.1
$C_{12}H_3Cl_7$	ND	<0.1		6	41	ND
$C_{12}H_2Cl_8$	ND	ND		ND	8	ND
$C_{12}H_1Cl_9$	ND	ND		ND	1	ND
$C_{12}Cl_{10}$	ND	ND		ND		ND

ND = non-detectable.

4. PRODUCTION

Commercial production of PCBs from the starting material benzene was begun in the 1920s by Swann Research, Inc., of Annington, Alabama, which referred to these products under the trade name Aroclor. PCBs were manufactured at that location by Swann Research, Inc., and its successor, Monsanto Chemical Company, until the plant was closed in 1971. Monsanto continued production at another plant at Sauget, Illinois, until 1977. The only other known manufacturer of PCBs is Geneva Industries of Houston, Texas, which manufactured PCBs from 1972 through 1974.

The domestic sale of Aroclor products peaked to 33,000 metric tons in 1970, and has declined since then due to restrictions on the use of PCBs (Hutzinger et al., 1974). PCBs were available commercially as mixtures (Aroclors) of 20 to 75 chlorinated biphenyls, and were marked according to the weight of chlorine contained in the mixtures. These commercial mixtures included Aroclors 1242, 1254, 1248, 1260, 1262, 1268, 1221, 1232, and 1016, in descending order according to domestic sales. In the year of peak production, 57% of the Aroclors produced were in the form of Aroclor 1242 (U.S. EPA, 1980a). The production in Japan and the annual consumption in Finland are estimated at 26 million pounds and 0.5 million pounds per year, respectively. PCB-1016 (41% chlorine) is a more recent product, and its sales prevailed for the period 1972-1976.

PCBs may be formed as side-products in other manufacturing processes involving the use of chlorinated benzene or biphenyl in the reaction step. For example, some of the trichlorobenzene used as a solvent in the manufacture of the dry pigment phthalocyanine blue is converted to PCBs during the reaction. PCBs formed can contaminate the pigment product at concentrations from a few parts per million to as much as 0.1%. Similarly, dichlorobiphenyl is formed in the manufacture of diarylide yellow pigments as a product of side reaction with the reactant dichlorobenzidine. The process of chlorinating water which contains biphenyl in a compound used as a dye carrier in dyeing polyester fibers can form PCBs as a side-reaction product which can contaminate the water. No natural sources of PCBs have been identified.

PCBs have been imported into the United States for use in various applications. Decachlorobiphenyl was imported from Italy for use as a wax filler in the investment casting industry until 1976. PCBs imported from France are used in mining machinery cooling systems. The percentage of imported PCBs over the total domestic sales for the period 1971 through 1975 in the United States is in the range of 1.6% to 2.7% (U.S. EPA, 1976b).

5. USES

Products containing PCBs have been used in agriculture and industry for decades. Their use is are mainly attributable to high chemical stability and physical properties desirable in certain applications. These properties include nonflammability, high dielectric constant, plasticizing capability, and ease of volatilization under heated conditions. Since 1930, PCBs have been extensively used as dielectric fluids in electrical transformers and capacitors, and have also been used for a variety of other purposes, including use in heat transfer and hydraulic systems, in the investment casting industry, and as plasticizers and solvents in sealants and adhesives. PCBs are also used as flame retardants in the manufacture of hard plastic products in which heat resistance is desired, as a dye carrier in carbonless copy paper, and as a plasticizer in paints.

Several published sources provide a comprehensive breakdown of uses for different types of Aroclors (Hutzinger et al., 1974; Nisbet and Sarofim, 1972; Versar, Inc., 1977). The most widely used Aroclors were 1242, 1248, 1254, and 1260. Aroclor 1016 was used after 1970 but in much smaller quantities than the four types mentioned above. A Monsanto marketing bulletin on PCBs, published in the 1960s, also described their possible use as gaskets and packing materials; as vehicles in graphic arts; as impregnation agents; as moisture-proof coatings; as wax substitutes; as de-dusting agents; in insecticides; in abrasives, lubricants, and cutting oils; in inks; in mastics; and in tank coatings (Monsanto Chemical Co., undated). A number of other uses have been patented.

In the United States, there are 17 companies that have used PCBs in the manufacture of askarel capacitors, and 13 companies that have used PCBs in the manufacture of askarel transformers. According to one study, in 1976 approximately 25 investment casting foundries (out of a total of 135 in the United States) used PCB-filled waxes in the manufacture of metal castings (U.S. EPA, 1976a).

In 1971, because of environmental concerns, the manufacturer voluntarily restricted the sale of PCB products for use only in "closed" systems, which include electrical transformers and capacitors with insulating fluids that contain PCBs. These two applications account for all of the current use of PCBs in the United States. The company was on a schedule to phase out production of all PCBs by 1979. The cessation of the production will reduce the amount of PCBs being released into the environment, but millions of pounds of PCBs are still being used in electrical insulation applications. The environmental contamination by existing PCBs, and their environmentally safe treatment or disposal, continue to be of concern.

6. DISPOSAL

A material balance performed on the amount of PCBs produced, sold, and purchased provides an estimate of the amount of PCBs lost or disposed of in the manufacturing process. The total estimated amount reported to have been disposed of or lost for the year 1974 is about 3.8 million pounds (U.S. EPA, 1976b), of which about 1.8 million pounds are estimated to have been land-disposed, and the rest to have been incinerated.

The 1.8 million pounds of PCBs in the land-disposed wastes generated from the manufacturing process amounts to only a small fraction of the total PCBs sent to land disposal facilities. The total land-disposed amount of PCBs for the year 1976 is reported to have been about 12 million pounds (U.S. EPA, 1976b). The most important source of PCB waste has been capacitors that have failed or become obsolete, or that are contained in obsolete equipment. Other PCB wastes include solid wastes from PCB manufacturing facilities and from operations using PCBs in non-electrical applications.

The data base for the WET Model, prepared by SCS Engineers (Undated), shows that PCB fluids containing 50% PCB-1254 sent to hazardous waste treatment, storage, and disposal facilities (TSDF) amount to about 4,500 tons per year. This waste competes for the capacity of TSDF regulated under Subtitle C of the Resource Conservation and Recovery Act. Water effluents from PCB production and first-tier use facilties are relatively small compared with the amounts being disposed of in landfills. Severe local impacts are evident by the discharge into rivers of these effluents. PCBs are now found in the sediments, water column, and biota in the rivers. A few examples of current PCB problems include the Hudson River and the New Bedford, Massachusetts, harbor. As a result of a strong tendency of PCBs to adsorb on sediments, and of sediment migration, PCB problems are identified farther downstream from the discharge points.

Twenty spills involving PCB products have been identified (U.S. EPA, 1976c). These spills occurred in transformer installations from trucks and railroad cars while they were en route to their destinations, and from leaking drums.

7. CHEMICAL AND PHYSICAL PROPERTIES

The chemical and physical properties of PCBs can be divided into two groups: (1) those relevant to the commercial and industrial use of PCBs, and (2) those that are needed in exposure evaluation and hence in developing media-specific safe level advisories for PCBs. The latter properties will be briefly summarized herein.

The widespread distribution of PCBs in the environment suggests that the major route by which PCBs are transported from treatment, storage, and disposal facilities is through the atmosphere in the form of volatilized vapor and adsorption on particulate matter. Vapor pressure is one of the important properties affecting volatilization. The available vapor pressure data for commercial Aroclors, as reported in the literature, have been compiled and are presented in this chapter. Vapor pressure, as distinguished from partial pressure or true pressure, refers to the maximum vapor-phase pressure achievable under equilibrium conditions at the soil-air interface.

Experimental data (U.S. EPA, 1980a) suggest that PCBs are strongly adsorbed on earth materials, including soil. PCBs adsorbed on soil, or present in the soil mixture, will be subject to ingestion if the contaminated sites are accessible to children or to adults with habitual pica. The bioaccumulation factor (in the food chain and in aquatic biota) is also an important physical parameter when there is a likelihood of PCB transport in water.

As pointed out previously, there are a number of congeners for each of the Aroclors. Thus, the properties listed herein for Aroclors represent averages over the various species that constitute the mixtures. The observation that environmental samples have contained more chlorobiphenyls with high chlorine levels than is characteristic of freshly manufactured Aroclors is attributable in large part to the possible metabolism and volatilization of lower chlorine species, coupled with enhanced sorption of species with more chlorine.

The more common types of Aroclors are shown in Table 4. Thirteen Aroclors were listed in a manufacturer's booklet (Monsanto Chemical Co., undated). These compounds range from oily liquids to white crystals and hard transparent resins, and generally have similar chemical and biological characteristics.

The properties and parameters commonly needed in estimating the environmental fate and transport of a given chemical are vapor pressure, solubility in water, soil-water partition coefficient, and bioaccumulation factor. These properties of PCBs, and other relevant properties, are shown in Table 4 (Burkhard et al., 1985; MacKay and Leinonen, 1975; Hutzinger et al., 1974; Monsanto Chemical Co., undated; Hwang, 1982; U.S. EPA, 1979a). Information on additional physical and chemical properties such as viscosity, softening points, and other factors, can be found in references authored by Hutzinger et al. (1974), Monsanto Chemical Co. (undated) and U.S. EPA (1980a).

The vapor pressure of PCBs and their solubility in water are low, and tend to decrease as the number of chlorine substitutions on the phenyl rings increase. Aroclors are soluble in most aliphatic and aromatic solvents, and are highly resistant to the action of strong alkali or acids, or high temperatures. Aroclors subjected to bomb tests are reported to have shown no evidence of oxidation (Hutzinger et al., 1974). PCBs have been shown to adsorb relatively rapidly and strongly to various materials, including soil, wood, plastic, and glass (Hutzinger et al., 1974).

Partition coefficients indicating a measure of partitioning under equilibrium conditions between PCBs at the interfaces of air-soil, air-water, water-soil media are important parameters in exposure analysis. Experimental data are scarce. Data for the distribution between air and water in the form of

TABLE 4. CHEMICAL AND PHYSICAL PROPERTIES OF PCBs

PCB	Molecular weight	K_{ow}	Specific gravity	Solubility[a] in water (mg/L)		Vapor press.(mmHg) at 25°C	Henry's law constant (atm. m^3/g mol)
PCB-1016 (Arochlor 1016)	257.9	24,000	1.182	0.42		$4x10^{-4}$	
PCB-1221	200.7	12,000	1.266	15.0		$6.7x10^{-4}$	
PCB-1232	232.2	35,000	1.380	1.45		$4.06x10^{-3}$	
PCB-1242	266.5	380,000	1.445	0.24		$4.06x10^{-4}$	$5.73x10^{-4}$[b]
PCB-1248	299.5	1,300,000	1.538	$5.4x10^{-2}$		$4.94x10^{-4}$	$3.51x10^{-3}$[b]
PCB-1254	328.4	1,070,000	1.620	$1.2x10^{-2}$	~0.03	$7.71x10^{-5}$	$8.37x10^{-3}$[c]
PCB-1260	377.5	14,000,000	1.646	$2.7x10^{-3}$		$4.05x10^{-5}$	$7.13x10^{-3}$[c]
PCB-1262			1.810				
PCB-1268			1.947				
PCB-1270			1.727				
PCB-2565			1.712				
PCB-4465			1.434				
PCB-5442			1.740				
PCB-5460							
2,2',5,5'-tetra-chloro-biphenyl				$4.6x10^{-2}$			
2,2',3,4,5,-penta-chloro-biphenyl				$2.2x10^{-2}$			

[a] Hutzinger et al., 1974; Monsanto Chemical Co., undated.
[b] MacKay and Leinonen, 1975.
[c] Hwang, 1982, and U.S. EPA, 1980c.

Bioaccumulation factor: 31,200 L/kg.

Soil-water partition coefficient (U.S. EPA, 1980a): 22 - 1938 L/kg.

Henry's Law constant and water and soil, exist for some selected Aroclors. Experimental data measuring the distribution of PCBs between air and soil are nonexistent. Estimates of air-soil partition coefficient can be calculated based on Henry's law constant and soil-water partition coefficient using one of several empirical relationships. The Henry's Law constant and soil-water partition coefficient, in turn, are dependent on water solubility and percent organic carbon in soil, respectively.

The Henry's Law constants shown in Table 4 are based on information in MacKay and Leinonen (1975) for PCB-1242, PCB-1248, and PCB-1260; and in a U.S. EPA research report (1980c) for PCB-1254. Burkhard et al. (1985) recently published a list of calculated Henry's Law constants for PCB-1242, PCB-1248, PCB-1254, and PCB-1260. The value for PCB-1254 is an experimental value obtained in the EPA laboratory in Cincinnati, Ohio, while others represent calculated values. A comparison of Henry's Law constants for PCB-1254 shows that the values in MacKay and Leinonen (1975) and Burkhard et al. (1985) differ by a factor of 10, while those in MacKay and Leinonen (1975) and the experimental EPA value (1980c) differ by a factor of 3. Since MacKay and Leinonen's value is closer to the experimental value, Henry's Law constants for PCB-1242, PCB-1248, and PCB-1260 are taken from MacKay and Leinonen (1975).

In the absence of experimental data, the soil-water partition coefficient K_d (cm^3 water/g soil), and the air-soil partition coefficient, K_{as} (g soil/cm^3 air) can be estimated from water solubility and percent organic carbon (%OC) in soil, using correlations presented by various researchers. For example, the values for the octanol-water partition coefficient, K_{OW}, can be used to estimate the values for the soil sorption coefficient based on soil organic carbon content, K_{OC} (cm^3 water/g organic carbon), and the bioconcentration factor (BCF) by the following formula:

$\log K_{OC} = 0.544 \log K_{ow} + 1.377$ (Kenaga and Goring, 1980) (1)

$\log K_{OC} = 1.00 \log K_{ow} - 0.21$ (Karickhoff, 1979) (2)

$\log BCF = 0.76 \log K_{ow} - 0.23$ (Veith et al., 1980) (3)

The K_d and K_{as} values then can be estimated by

$$K_d = K_{OC} \left(\frac{\%OC}{100}\right) \tag{4}$$

$$K_{as} = \frac{H}{K_d} \tag{5}$$

where H represents Henry's Law constant. Since the common unit for H is given in atm m^3/g mol, a conversion factor of 41 ($=1/2.44 \times 10^{-2}$) should be multiplied in the right-hand side of Eq. (5) when the units for K_{as}, H, and K_d are g soil/cm^3 air, atm m^3/g mol, and cm^3 water/g soil, respectively. The multiplication of K_{as} by the concentration of PCBs in soil will provide the concentration of PCBs in the air phase above contaminated soil of interest under equilibrium conditions. It should be recalled that the air-soil partition coefficient, K_{as}, has the unit of g soil/cm^3 air. This is equivalent to the ratio of the air-phase to soil-phase concentration, or (mg/cm^3 air)/(mg/g soil). The estimation of K_{as} requires the knowledge of Henry's Law constant and the soil-water partition coefficient as given by Eq. (5).

A listing of solubilities of each chlorinated biphenyl is shown in Table 5 (U.S. EPA, 1976c).

There has been much speculation as to the possible role of photochemical reaction in the environmental decay of PCBs. The results of a study using mercury vapor (UV) sources (U.S. EPA, 1976c) have been difficult to extrapolate to environmental conditions because the radiation wavelength is not within the spectrum of solar radiation at the surface of the earth. More recent experiments have been reported using a light source more closely approximating the spectral distribution of solar radiation, but the values for the photochemical reaction constants are not available.

The Monsanto Chemical Company has reported vapor pressure data only for high-temperature conditions for Aroclors 1242, 1248, 1254, and 1260 (Monsanto Chemical Co., undated). The temperature used in presenting the data ranges from 150°C to 300°C. These vapor pressure values may be extrapolated to the temperatures which are of common environmental concern, but their accuracies would be doubtful.

An EPA report presents kinetic data obtained from biodegradation experiments using water-soluble Aroclor 1242 (U.S. EPA, 1980a). The rate constants are presented for biphenyl compounds with up to the three chlorine substitutions present in Aroclor 1242. The data clearly show that many of the chlorinated biphenyls with four chlorine substitutions do not biodegrade after 48 hours of degradation run. Inferring from the compositions of Aroclors 1242 and 1254 as given in Table 3, it is conceivable that Aroclor 1242 may biodegrade to some extent because it contains a substantial amount of chlorinated biphenyls with two and three chlorine substitutions. It appears that biodegradation of Aroclor 1254 would be insignificant or may not occur because most biphenyl components have four or more substituted chlorine atoms.

PCBs have several properties which make them toxic in the environment. In addition, they can significantly bioaccumulate and concentrate in the fatty tissues of all organisms. For example, the PCB concentration in resident fish is often many times higher than that in the surrounding water. PCBs are chemically stable compounds that are able to persist in the environment for long

TABLE 5. SOLUBILITY OF CHLOROBIPHENYLS IN WATER

Compound	Solubility mg/L (ppm)
Monochlorobiphenyls	
2-	5.9
3-	3.5
4-	1.19
Dichlorobiphenyls	
2,4-	1.40
2,2'-	1.50
2,4'-	1.88
4,4'-	0.08
Trichlorobiphenyls	
2,4,4'-	0.085
2',3,4-	0.078
Tetrachlorobiphenyls	
2,2',5,5'-	0.046
2,2',3,3'-	0.034
2,2',3,5'-	0.170
2,2',4,4'-	0.068
2,3',4,4'-	0.058
2,3',4',5-	0.041
3,3',4,4'-	0.175
Pentachlorobiphenyls	
2,2',3,4,5'-	0.022
2,2',4,5,5'-	0.031
Hexachlorobiphenyl	
2,2',4,4',5,5'-	0.0088
Octachlorobiphenyl	
2,2',3,3',4,4',5,5'-	0.0070
Decachlorobiphenyl	0.015
4,4'-Dichlorobiphenyl	
+Tween 80 0.1%	5.9
+Tween 80 1%	>10.0
+Humic acid extract	0.07

periods. Impurities in commercial PCBs could amplify the PCB problem because of their similarity in chemical structure and toxicity (Monsanto Chemical Co., undated).

8. ENVIRONMENTAL DISTRIBUTION

The release of PCBs into the environment through disposal on or in land, and through effluent discharges into waterways, together with their high attenuation characteristics and long half-life, has resulted in detectable levels in ambient air, soil, rivers, sediments, and in tissues of fish, wildlife, cattle, poultry, and a large portion of the human population. Measurable amounts of PCBs have been found in Antarctic ice, showing that atmospheric transport over long distances does occur (U.S. EPA, 1976b). Monitoring shows that the soils in the rural and urban areas where there is no record of PCB disposal or contamination, contain detectable amounts of PCBs (U.S. EPA, 1976c). One study estimates that 70% of the PCB load to Lake Michigan is through atmospheric transport (University of Wisconsin, 1980).

The results of soil sampling show that PCBs are more prevalent in urban soil than in agricultural soil. Data indicate that PCBs are rarely detected in agricultural soil, while urban soils showed PCB contaminations up to about 12 μg/g soil, with averages ranging from 0.01 to 0.21 μg/g (U.S. EPA, 1976c; Carey, undated). Sixty-three percent of the soil samples showed detectable PCB levels. The most prevalent PCBs in soil were Aroclor 1254, and, to a lesser extent, Aroclor 1260.

The PCB concentrations in air samples over Lake Michigan taken during 1977 (University of Wisconsin, 1980) were lower than in those taken in the urban portion of Milwaukee. The main components were identified as Aroclors 1242 and 1254, while the particulate-phase PCBs contained Aroclor 1260 in some instances. The average concentration of PCBs in the air over Lake Michigan was 0.87 ng/m^3 (0.44 to 1.33 ng/m^3). The concentrations of PCBs in the particulate samples were similar to those in the air samples. Air samples taken in later years from Lake Michigan showed an average concentration of 1 ng/m^3. These concentrations are lower than those reported in the ambient air in the continental United States.

The ambient air concentrations of PCBs for urban Chicago averaged 7.7 ng/m^3. The average composition in the air sampled was 86% Aroclor 1242, 13% 1254, and 1% 1260. The particulate-phase PCBs had a slightly different composition for the same Aroclors. The ambient air in Milwaukee showed an average PCB concentration of 2.25 ng/m^3.

9. ENVIRONMENTAL FATE AND TRANSPORT

PCBs have been found in samples of air, water, soil, sediments, fish, birds, and mammals (including humans) all over the world (U.S. EPA, 1980a). Once released into the environment, PCBs persist and collect in animal tissues. Environmental problems caused by PCBs were largely unreported until 1966, when PCB contamination of fish, eagles, and humans was detected. The best-documented incident concerning the effects of ingested PCBs on humans is the case that occurred in Yusho, Japan, in 1968 (U.S. EPA, 1980a). Several other cases have also been reported (U.S. EPA, 1980a, U.S. EPA, 1976c).

Sediments containing PCBs have been reported in rivers, estuaries, and harbors (U.S. EPA, 1981b), in concentrations ranging from 20 to 50,000 µg/g. Leaching of PCBs could occur, although it will be constrained by the low solubility limits. Once PCBs dissolved in water enter the soil medium, it is possible that further migration will be severely retarded in view of the high soil-water partition coefficients. The retardation factors calculated from these coefficients can be used to simulate the arrival time and concentration decrease in groundwater. This will be further explained later.

Despite their low vapor pressures, PCBs can volatilize into the atmosphere. The migration of PCBs through air is considered to be one of the basic mechanisms by which the ubiquitous presence of PCBs occurs in nature (U.S. EPA, 1980a). The New York State Department of Environmental Conservation (NYSDEC) analyzed samples of PCB-contaminated air at several localities. These analyses are shown in Table 6 (NYSDEC, 1979; U.S. EPA, 1981b). Concentrations of PCBs in the ambient air as high as 300 µg/m^3 were reported at the disposal site. The average values ranged from 130 to 0.3 µg/m^3. Concentrations of suspended particulates in the air in the vicinity of dump sites were also monitored by

TABLE 6. PCB MONITORING IN AMBIENT AIR BY NYSDEC

Site	Max. PCB conc. ($\mu g/m^3$)	Average PCB conc. ($\mu g/m^3$)
Caputo dump	300	130
Fort Miller dump	35	24
Remnant area	10	9
Moreau site	15	5.6
Buoy 212 site		0.7 (one sample)
Old Moreau Site (Summer 1979)		0.3

NYSDEC, using high-volume air samplers (NYSDEC, 1979; U.S. EPA, 1981b). The geometric annual mean concentration of particulate matter was 36 to 63 $\mu g/m^3$, while the 24-hour averages were 71 to 144 $\mu g/m^3$. The amount of PCBs adsorbed to the collected suspended particulates was not reported.

Based on mass-transfer models, MacKay and Leinonen (1975) calculated the half-lives of PCBs present in solution in a water column of 1 m depth. Half-lives provide some indication of how fast a compound can volatilize from solution. The half-lives for Aroclor-1242, Aroclor-1248, Aroclor-1254, and Aroclor-1260 are reported to be 12.1 hr, 9.5 hr, 10.3 hr, and 10.2 hr, respectively. Half-lives will be longer when depths are greater than 1 m. The calculated half-lives are for evaporation from a calm, liquid surface. Turbulence provides exchange of contaminants between the surface layer and the bulk of the water column. This exchange results in increased emission rates, thus providing shorter half-lives.

In addition to the importance of attenuation mechanisms when PCBs interact with soil, biodegradation is also suggested as a potentially important mechanism. Biodegradation studies using pure and mixed microbial cultures, and the resulting metabolic changes in PCB compounds, have been summarized by Hutzinger et al. (1974) and Hwang (1982). Photochemical degradation of PCBs in the atmosphere is also of interest, since a number of pesticide compounds have been shown to break down through the photochemical route. However, very little information is available in the literature to determine the extent of PCB degradation in the atmosphere.

The safe disposal and treatment of PCBs discarded after their use in electrical applications is a matter of great concern with regard to human health. Incineration techniques are frequently applied to PCB material at elevated temperatures and high residence times. Several experiments involving pyrolysis of commercial PCBs have been reported (Buser and Rappe, 1979). The PCBs used in the pyrolysis experiments included tetrachlorobiphenyl, pentachlorobiphenyl, hexachlorobiphenyl, heptachlorobiphenyl, and octachlorobiphenyl. The analysis of the pyrolysis residues showed the presence of chlorinated furan compounds. However, the researchers concluded that the formation of furan compounds is the result of uncontrolled burning of PCBs, and that the emission of these compounds can be reduced by controlling the burning process.

The high-temperature combustion of PCBs, such as in the case of transformer fires, results in the formation of polychlorinated dibenzofurans (PCDFs) and other toxic compounds. In an experiment studying conditions favoring the formation of polychlorinated dibenzodioxins (PCDDs), researchers found that the optimum conditions for the formation of PCDFs are a temperature of near 675°C at a residence time of 0.8 seconds or longer, with 8% excess oxygen (Midwest Research Institute [MRI], 1984). No conditions for the formation of PCDDs are represented. The report states that detection of PCDDs was occasional and at low levels.

10. TOXICOLOGY

The advisory levels for PCB cleanup presented in this document (i.e., the permissible PCB soil contamination levels) are health-based values. These advisories are derived through calculations which first estimate human risk-specific (cancer end point) or acceptable intake (AI) (noncancer end point) levels, and then determine the exposure rates which will effect these intake levels. Risk-specific doses are derived for the cancer end point, and a 10-day AI level is derived for an approximate 10- to 30-day exposure considering only noncancer effects. A detailed assessment of the available cancer and noncancer health effects data for PCBs is presented in Appendix D. Only a brief overview and major issues will be presented here.

The determination of risk-specific intake levels is accomplished through a mathematical process which makes use of a cancer potency factor and a reflected risk level or levels to estimate the intake level that would correspond to such risk levels. Cancer potency factors for PCBs have been determined through an exhaustive analysis of animal studies. Values have been calculated by ORD (EPA, 1980b) to be 4.34 $(\text{mg/kg}\cdot\text{day})^{-1}$ and by OTS (EPA, 1985b) to be 3.57 $(\text{mg/kg}\cdot\text{day})^{-1}$. An average of these values, or 4.0 $(\text{mg/kg}\cdot\text{day})^{-1}$ is used in the calculations presented in this document. A discussion of the data and methods used to estimate cancer potency factors for PCBs is included in Appendix D. The determinations made by ORD and OTS are both based on the same animal study (Kimbrough et al., 1975), with only slightly different assumptions being incorporated.

A noncancer AI level was derived for PCBs during the preparation of this report. It must be emphasized that this AI ignores the cancer end point and is based on toxicity other than cancer. The 10-day AI level of 100 µg/day for a child and 700 µg/day for an adult, derived for use in this document, is based on feeding studies with rabbits and rats in which a NOAEL for decreased reproductive rate, and effects on thyroid and liver, were evaluated. These studies are described briefly below.

Villeneuve et al. (1971) found increased incidences of fetal death, resorptions, and abortions at 12.5 mg/kg/day of Aroclor 1254 in rabbits when exposed on days 1 through 28 of pregnancy. A dose of 1.0 mg/kg/day appeared to be without effect. Collins and Capen (1980a, b, c) in a series of studies on thyroid effects in rats, determined that 50 µg PCB per g of diet (~ 2.5 to 5.0 mg/kg/day) for 4 weeks was associated with clearly defined adverse effects, but that doses of 5 µg PCB per g of diet (~ 0.25 to 0.5 mg/kg/day) were not. Carter (1983) demonstrated liver hepatomegaly in rats at doses of 20 µg Aroclor 1254 per g of diet (~ 2 mg/kg/day) for 14 days; such an effect, in the absence of other signs of toxicity (e.g., fatty infiltration of the liver), might not be considered adverse. Grant and Phillips (1974) observed increased liver weights in rats at doses as low as 5 mg/kg/day Aroclor 1254 given in corn oil for 7 consecutive days. Collectively, these studies indicate that the experimental threshold for adverse effects of Aroclor 1254 in studies of 30 days' duration or less is at or near a dose of 1 mg/kg/day. Thus, it seems reasonable to use this latter dose, a No Adverse Effect dose, as a basis for health advisories for Aroclor 1254 for short exposure durations.

11. EXISTING STANDARDS AND GUIDELINES

The 1968 incident in which toxic symptoms were experienced by Japanese people exposed to contaminated cooking oil gave rise to a great deal of concern in the United States with regard to hazardous chemicals. The U.S. Food and Drug Administration (FDA) started sampling foods for possible contamination by PCBs in 1969, and detected levels of PCBs in fish from the Great Lakes, in milk caused by use of materials containing PCBs, and in chickens as a result of the existence of PCBs in the feed. The temporary tolerance levels for residues of PCBs proposed by FDA became effective June 29, 1979 (U.S. FDA, 1984).

In the early 1970s, EPA proposed the establishment of criteria for PCBs being discharged in industrial effluents, but has not so far issued effluent limitations concerning PCBs.

The Occupational Safety and Health Administration (OSHA) adopted standards for PCB exposure by industrial workers. Subsequently, the National Institute for Occupational Safety and Health (NIOSH), after their extensive assessment, recommended lowering the allowable concentration of PCBs in the workplace. However, OSHA has not acted on this recommendation. The New York State Department of Health issued an interim guideline for the allowable ambient air concentration of PCBs to which individuals may be exposed during the duration of a PCB reclamation project planned for the Hudson River (NYSDEC, 1979).

The EPA promulgated regulations relating to manufacture, processing, distribution in commerce, use, diposal, storage, and markings of PCBs and PCB items. The regulations originally became effective May 31, 1979, and were later amended. A complete presentation of the effective regulations can be found in the latest edition of 40 CFR Part 761 (U.S. EPA, 1984b). The PCBs referred to in these regulations include any chemical substances or their mixtures containing concentrations of chlorinated biphenyls of 50 ppm or greater. The regulations pertain to prohibitions on manufacturing, processing, distribution in commerce, and use, and specifically apply to PCB incinerators, chemical waste landfills disposing of PCBs, transformers, pigments, electrical and heat transfer equipment, natural gas pipeline compressors, microscopy mounting medium, capacitors, PCB containers, and hydraulic systems (U.S. EPA, 1984b).

The PCB standards and guidelines for numerical limitations of PCBs in food, drinking water, and ambient air existing at the present time are shown in Table 7. Because of the complicated nature of the EPA's regulations promulgated under TSCA, these regulations are not presented in tabular form.

TABLE 7. EXISTING PCB STANDARDS AND GUIDELINES

Exposure pathways	Maximum allowable PCBs
Food (FDA standard)[a]	
Milk fat and dairy products	1.5 μg/g (ppm)
Poultry	3.0 μg/g (ppm)
Eggs	0.3 μg/g (ppm)
Fish and shellfish	2.0 μg/g (ppm)
Finished animal feed	1.0 μg/g (ppm)
Drinking water (New York State)	1.0 μg/L (ppb)
Ambient air	
Populated areas (New York State guideline)[b]	1.0 μg/m^3
Workplace (OSHA standard)	500 μg/m^3
Work site (NIOSH guideline)[c]	1.0 μg/m^3

[a] U.S. FDA, 1984.
[b] New York State Department of Health, 1981.
[c] NIOSH, 1977.

12. EXPOSURE ASSESSMENT METHODOLOGY

The presence of PCBs in environmental media poses a potential health risk to humans from the following sources of intake:

- ingesting contaminated soil
- inhaling contaminated air
- ingesting contaminated food
- drinking contaminated water
- dermal absorption of PCBs in contact with skin

Other exposure pathways affecting ecological communities, such as phytotoxicity to plants, may also need to be considered. If multiple-route exposures are possible, the concentrations allowable for a single-route exposure should be adjusted to meet the acceptable levels of acute and chronic health effect exposures from all sources of intake. The amounts of each medium subject to human intake used in this analysis are as follows: daily intake of drinking water, 2 L/day; daily inhalation of air, 20 m^3/day (U.S. EPA, 1984b).

Acute effects result from short-term or long-term intakes. Carcinogenic effects are normally treated as resulting from lifetime intakes. Safe levels of PCBs in soil corresponding to 1-day and 10-day acceptable intakes should be based on consideration for preventing acute health effects from short-term and longer-term exposures. Levels of PCBs in soil corresponding to acceptable intakes for long-term effects can be derived from the acceptable daily intake (ADI) based on long-term health studies for acute effects, or from the carcinogenic potency slope based on lifetime exposure for carcinogenic effects.

The long-term risk level for ingestion of contaminated soil over a 70-year lifetime exposure can be obtained by

$$\text{Risk} = \sum_{t=0}^{69\ \text{yrs}} \frac{(GI)\ (SM)\ Co\ e^{-kt}\ (POT)\ (IR)}{(BW)\ (LT)} \tag{6}$$

where Risk = lifetime risk; GI = gastrointestinal tract absorption of PCBs; SM = exposure frequency over a lifetime; Co = initial concentration of PCBs in soil; k = biodegradation constant (1/day); POT = potency slope factor (mg/kg/day)$^{-1}$ for PCBs; IR = daily ingestion rate of soil; BW = body weight (70 kg for adults and 10 kg for children); and LT = exposure time over a lifetime (70 years). If the contaminant undergoes biological or chemical degradation in soil, and follows first-order kinetics in its disappearance under isothermal conditions, the contaminant concentration will change as a function of time according to C_0e^{-kt}. The summation in Eq. (6) is necessary in order to add all the risks associated with the daily dosage over a lifetime. The initial concentration of PCBs in the soil is calculated at an assumed lifetime risk according to Eq. (6). A computer is convenient to use to sum all the daily risks. The soil ingestion scenario will be applicable to sites which are readily accessible, especially by children. Since the population is living on or near the site, the exposure to PCBs due to inhalation of contaminated air cannot be neglected. In order to account for the inhalation exposure in determining the allowable PCB levels in soil due to the combined routes of ingestion, inhalation and dermal absorption, the ambient air concentrations at the exposure points are needed. The concentration of PCBs in the vapor phase is the result of the volatilization of the PCBs from the contaminated soil and their dilution by winds. The dilution factor for ambient air concentrations can be defined as

$$D = C_a/C_{as} \tag{7}$$

where C_a ($\mu g/m^3$) = the ambient air concentration at an exposure location, and C_{as} ($\mu g/m^3$) = the concentration of PCBs in air at the soil surface where the emission occurs. The value of C_{as} continuously increases as the PCB concentration in soil increases, until the concentration of PCBs in the air phase corresponds to that of PCB vapor pressure. Beyond this point, a further increase in PCB concentration in soil will have a minimal effect on the volatilization rate.

Once exposure pathways are identified, exposure evaluation requires information on the levels of concentration to which a given target population may be exposed. Each pathway may require route-specific evaluation. A general method for estimating exposures for contaminated sites will be described first. The method can be simplified by eliminating those pathways that are unimportant or unrelated in the evaluation of PCB advisories. The relevant assumptions for the simplification are described below.

12.1 Estimation of Exposures for Contaminated Sites

The combined human intake of contaminants from all exposure pathways should not exceed the acceptable intake (AI, in mg/day) needed for preventing adverse effects from short-term and lifetime exposures. The intake from an individual route when soil is contaminated can be expressed quantitatively as follows:

i) Intake by soil ingestion (mg/day):

$$I_1 = (C_s)(IR \times 10^{-3})(GI)(SM)(F) \tag{8}$$

The term $(C_s)(IR)$ in Eq. (8) represents the daily amount of a contaminant ingested resulting from soil ingestion, in µg/day, because C_s represents the contaminant concentration in soil (μg/g) or ppm (both units are equivalent), and IR is the soil ingestion rate (g/day) and GI is defined in Eq. (6). The factor 10^{-3} is needed to convert the unit from μg/day to mg/day. The fraction of the ingested contaminants that will enter human organs and systems to cause toxicity is given as GI. An individual may not always be present on the contaminated site over his lifetime. The frequency factor of exposure over a lifetime, SM, represents the fraction of a lifetime that an individual will be exposed to the contaminants under consideration. The factor F is necessary because soil ingestion only occurs during childhood (1 to 5 years of age), and the weight of the human body changes from childhood to adulthood.

ii) Intake by air inhalation of volatilized contaminants (mg/day):

$$I_2 = (K_{as})(C_s)(D)(IH \times 10^3)(ABA)(SM) \tag{9}$$

In Eq. (9), K_{as}, D, and IH represent the soil-air partition coefficient, given by Eq. (6), g soil/cm^3; the extent of dilution as given by Eq. (7); and the average daily inhalation rate of ambient air (m^3/day), respectively. The term $(K_{as})(C_s)(D)(10^3)$ represents the ambient air concentration of a pollutant at the exposure location in mg/m^3 when K_{as} and C_s are given in g soil/cm^3 and mg/kg, respectively.

The determination of the ambient air concentration at an exposure location, $(K_{as})(C_s)(D)(10^3)$, or the dilution factor in the term, requires the estimation of transient emission rate, and the use of dispersion modeling. The emission rate will not only be transient, but it will also be retarded by soil or equivalent cover material. This phase of the problem requires solution of a partial differential equation, as described in the Section 16.

If the concentration of PCBs in soil is at or above saturation conditions, under which the air phase concentration of PCBs is equal to the vapor pressure concentration for a particular Aroclor or a mixture of Aroclors, the further increase in C_s will not increase the ambient air concentration, assuming that other factors, such as temperature, remain constant. Therefore, the daily intake by inhalation remains constant above the concentration of PCBs in soil providing saturated air concentration. This concentration of PCBs in soil, or the saturated concentration in soil for air inhalation, will be denoted by C_{sm}.

iii) Intake by dermal absorption (mg/day):

$$I_3 = (C_s)(CR \times 10^{-3})(ABS)(SM) \qquad (10)$$

There are many occasions when children playing in the yard or adults working in the garden will come in direct contact with contaminated soil. Dermal contact does not necessarily constitute adverse exposure. The contaminant needs to be systemic to be absorbed into the human body and to exert toxicity. In Eq. (10), the term $(C_s)(CR)$ represents the contaminant contact rate with skin in μg/day since C_s is in μg/g (=ppm) and CR is the dermal contact rate of soil in g/day. The factor 10^{-3} is used to convert the contact rate from μg/day to mg/day, and SM will be 1 when the short- or longer-term (10-day) exposure is estimated, and will be between 0 and 1 when the lifetime exposure is estimated.

iv) Intake by drinking water (mg/day):

$$I_4 = (C_w)(IW)(SM) \tag{11}$$

In Eq. (11) it is assumed that the contaminant in drinking water is completely absorbed into the human body at the average daily water consumption rate of IW or the absorption fraction is 1. In order to relate the contaminant concentration in groundwater, C_w, to the contaminant concentration in soil, C_s, a fate and transport model can be used to estimate the concentration in the leachate entering groundwater, or

$$C_w = C_L/f_g, \text{ mg/L} \tag{12}$$

where f_g represents a functional relationship describing contaminant transport in groundwater. This function should be selected to suit the most appropriate conditions for the system. The leachate concentration, C_L, referring to the contaminant concentration in liquids just before entering groundwater, should not be confused with the contaminant concentration in groundwater, which results from mixing of the leachate with groundwater. Also, care should be exercised in using groundwater transport models, because some models will treat the leachate concentration as a boundary condition, while others require the contaminant concentration in groundwater as a boundary condition, which should be obtained by groundwater monitoring. When the units of C_w and C_L are all in mg/L, then the function f_g becomes dimensionless. Most leachate from hazardous waste land disposal sites may enter groundwater over a finite surface area, favoring area source models for simulating pollutant transport in groundwater.

There is no reliable method of predicting the leachate concentration from the contaminant concentration in soil, or vice versa. For the exposure evaluation, an equilibrium relationship vetween soil and leachate will provide a first approximation. Monitoring data can also be used relating the concentrations between leachate and soil. An equilibrium condition can be written as

$$C_s = (K_{LS})(C_L),\ \text{mg/kg} \tag{13}$$

where K_{LS} is a partition coefficient in (mg/kg)/(mg/L). Eqs. (11), (12), and (13) are combined to get

$$I_4 = \frac{C_s}{(f_g)(K_{LS})}(IW)(SM) \tag{14}$$

When the equilibrium condition is not appropriate, it can be modified to include transport processes between the soil and leachate.

v) Intake by fish ingestion (mg/day):

At the average daily fish consumption rate of IF (kg/day), and under the assumption of complete absorption of the contaminant associated with the consumption of fish, the exposure can be estimated as

$$I_5 = (C_F)(IF)(SM) \tag{15}$$

where C_F is the contaminant concentration in fish. The use of the bioconcentration factor BCF, (mg/kg fish)/(mg/L water), to relate pollutant concentrations in fish and water, gives

$$I_5 = (BCF)(C_w)(IF)(SM) \quad (16)$$

where it is assumed that contaminants are present in water in dissolved form and that bottom sediments or benthal deposits on which pollutants may be adsorbed are not directly swallowed by fish. Under the condition of equilibrium between the pollutant-containing soil and leachate which is generated from the soil, substitution of Eqs. (12) and (13) into Eq. (14) results in

$$I_5 = \frac{C_s}{(f_g)(K_{LS})}(BCF)(IF)(SM) \quad (17)$$

The transport functions, f_g, in Eqs. (14) and (17) may assume distinct mathematical descriptions, because one pertains to transport in groundwater and the other to that in surface water.

vi) Intake by inhalation of contaminants adsorbed on particulates (mg/day) may be expressed by

$$I_6 = (C_p)(IH)(C_s \times 10^{-9})(ABP)(SM) \quad (18)$$

Contaminant-containing soil can be airborne by blowing winds. In addition, toxic substances volatilized from contaminated soil can be adsorbed on particulate matter present in the ambient air. Exposure to contaminants occurs because of inhalation of air containing these particulates. The exposure location could be distant from the source of emission, or in the vicinity of the emission source. Exposure concentrations will change accordingly. Another form of exposure relates to inhalation of air containing particulate matter on which volatile constituents are adsorbed. The intake rate can be estimated based on the concentration of contaminants in wind-dispersed soil or on particulate matter, C_s μg/g (=ppm), and the concentration of the particulates in the ambient air, C_p μg/m^3, as shown in Eq. (18). The absorption fraction, ABP, is used because contaminants present in or on soil (or particulate matter) may be bound on the solid material, reducing the contaminant's absorption rate. Finally, the factor 10^{-9} is a conversion factor to make the units consistent.

vii) Intake by ingestion of vegetables (mg/day):

The intake rate due to ingesting IV kg/day of vegetables, plants, or agricultural products containing C_v mg/L of contaminants will be

$$I_7 = (C_v)(IV)(SM) \tag{19}$$

If it is assumed that equilibrium is established between the contaminant concentrations in plant and soil, then the exposure can be modified as:

$$I_7 = (K_{sv})(C_s)(IV)(SM) \tag{20}$$

where K_{sv} is a partition coefficient defined as contaminant concentration in plant/total contaminant concentration in soil (mg/kg plant)/(mg/kg soil).

viii) Intake by ingestion of food meat:

The contaminant intake at consumption rate of IM (kg/day) of meat containing C_m (mg/kg) of pollutant is

$$I_8 = (C_m)(IM)(SM) \tag{21}$$

Here again, an equilibrium relationship is assumed between the contaminant concentrations in the animal body and plants. Therefore, the intake rate due to meat consumption is

$$\begin{aligned} I_8 &= (K_{vm})(C_v)(IM)(SM) \\ &= (K_{vm})(K_{sv})(C_s)(IM)(SM) \end{aligned} \tag{22}$$

where K_{vm} and K_{sv} are the partition coefficients used to describe pollutant distribution between meat and vegetables, and the partition between vegetables and soil, respectively.

12.2 Determination of Permissible Pollutant Levels in Soil

The total intake from all possible exposure pathways is set equal to the acceptable intake (AI) for short-term and chronic health effects; or

$$AI = I_1 + I_2 + I_3 + \cdots \tag{23}$$

Eq. (23) can be solved for permissible contaminant levels in soil corresponding to each acceptable intake. It is possible that some exposure pathways occur independently of others. For example, a residence which is located on a contaminated site may use drinking water from a clean public water treatment system, and may thus be free of contaminants found on the site. It is also possible that domestic animals are not raised for food consumption on the contaminated site under consideration. Under such circumstances, all exposure pathways need not be considered. If exposure pathways of significant concern are related to soil ingestion, inhalation of contaminated air, or dermal contact with soil, as is the case for development of PCB advisories, Eqs. (8), (9), and (10) can be added to solve for C_s,

$$C_s = \frac{(AI)(1000)}{[(IR)(GI)(F) + (K_{as})(D)(IH)(ABA \times 10^6) + (CR)(ABS)]SM} \tag{24}$$

The emission rate is limited by the air phase concentration in equilibrium with the contaminant concentration in soil. Once the contaminant soil concentration reaches the level at which the vapor phase concentration in equilibrium with the soil is at the vapor pressure concentration, a further increase in contaminant concentration in soil ($C_s > C_{sm}$) does not increase the emission rate. At or above this concentration, the ambient air concentration remains constant regardless of the concentration of the contaminant in soil. Under such conditions, C_s in Eq. (9) is no longer a variable, and therefore Eq. (24) does not apply. This situation can be remedied by considering the intakes by the individual route of exposure at a constant value of C_s [$C_s = C_{sm}$ in Eq. (9)] for inhalation exposure, and solving for C_s. The form of the equation will be slightly different from that for Eq. (24).

$$C_s = \frac{(AI)(1000) - (K_{as})(C_{sm})(D)(IH \times 10^3)(ABA)(SM)}{[(IR)(GI)(F) + (CR)(ABS)]SM} \tag{25}$$

12.3 Incorporation of Time-Varying Parameters

The body weight of a human constantly changes until maturity. The calculation of AIs from the safe dose level (SL) given in mg/kg•day requires an assumption of body weight. For rigorous treatment, the estimation of lifetime exposure should take into account changes in body weight. In this case, it is convenient to work with SL instead of AI for exposure calculations. For carcinogens with a potency value at POT $(\mathrm{mg/kg \cdot day})^{-1}$, the equivalent SL at an assumed risk level, R (such as 10^{-6}, etc.), can be obtained by

$$(SL)eq. = \frac{R}{POT}\ , \ \mathrm{mg/kg \cdot day} \tag{26}$$

The risk level shown represents an upper-bound estimate. An upper-bound estimate of risk of 10^{-6}, for example, means that upon lifetime exposure to a contaminant, a person experiences an increased maximum risk of developing cancer in a probability of 1 in one million.

Snyder (1975) presented data on the change of body weight as a function of age. A regression analysis on Snyder's data for average male weight provides the following relationship.

$$BW = 3.14 + 3.52\ (\mathrm{age}),\ \mathrm{kg\ for\ age\ 0 - 18\ yr} \tag{27}$$

$$BW = 70,\ \mathrm{kg\ for\ age\ greater\ than\ 18\ yr} \tag{28}$$

To obtain the daily exposure averaged over an individual's lifetime, intake rates given by Eqs. (8) - (10), (10), (17), (18), (20), and (24) should be divided by the body weight, and the daily intake per unit body weight should be averaged by summing the total intake per unit body weight over the period during which exposure occurs and dividing the result by LT. For purposes of illustration, Eqs. (8) and (9) are repeated below:

i) The average daily exposure by soil ingestion per unit body weight in mg/kg•day can be determined as

$$\frac{I_1}{BW} = \sum_{1\ \text{day}}^{25550\ \text{days}} \frac{(C_0 e^{-kt})(IR \times 10^3)(GI)(SM)}{(BW)(LT)} \qquad (29)$$

Again, in Eq. (29) [also in in Eq. (30)], the contaminant present in soil is assumed to disappear by biodegradation and other reactions, according to first-order kinetics. Other processes affecting the concentration in soil are considered in the exposure analyses for individual pathways.

ii) The average daily exposure by inhalation of volatilized contaminants in mg/kg•day is calculated from

$$\frac{I_2}{BW} = \sum_{1\ \text{day}}^{25550\ \text{days}} \frac{(K_{as})(C_0 e^{-kt})(D)(IH \times 10^3)(ABA)(SM)}{(BW)(LT)} \qquad (30)$$

Similar expressions can be written for other exposure pathways. For conservative contaminants, the term C_0e^{-kt} in Eqs. (29) and (30) can be replaced by C_s. The total dose from all exposures should not exceed SL, or $(SL)_{eq.}$

$$SL = \frac{I_1}{BW} + \frac{I_2}{BW} + \frac{I_3}{BW} + \ldots \qquad \text{for noncarcinogenic effects} \qquad (30)$$

$$(SL)_{eq.} = \frac{I_1}{BW} + \frac{I_2}{BW} + \frac{I_3}{BW} + \qquad \text{for carcinogenic effects} \qquad (32)$$

As before, Eq. (31) or (32) can be solved for the permissible concentrations in soil, C_s. From Eqs. (8) and (29), one can solve for the factor F for use in Eq. (8). The use of LT = 25550 days, and the assumption that soil ingestion occurs during ages 1 through 5 (t = 365 to 1825 days), yield F = 0.323. The factor F does not depend on the soil ingestion rate. Eqs. (8) and (29) use Eqs. (28) and (27), respectively, for BW.

12.4 PCB Advisory Evaluations

Under normal conditions, significant soil ingestion is limited to children (Lepow, 1975). Although very limited information is available on the ranges of age subject to soil ingestion, one investigation presented a case study of an adult with a history of habitual eating of garden soil, which may have been associated with a pica illness (Wedeen et al., 1978). The fraction of soil contaminant absorbed by humans is dependent upon the type of compound and its soil contaminant adsorption characteristics, and is generally smaller than that which can be expected when contaminants are present in food or drinking watder.

PCBs can be removed from surface water, leaving it suitable for drinking. Well water that comes from ground water could be a direct source of drinking water. The location of the drinking water exposure does not necessarily have to be at the site of the contamination. It is assumed that the population which may be subject to PCB contamination in drinking water is remote from the PCB-contaminated sites, and the allowable water concentration is separately calculated on the basis of not eating contaminated soil and not inhaling contaminated air in the immediate vicinity of the site. The water concentration for a single-route exposure can be calculated as

$$C_w = \frac{AI}{2\ L/day} \qquad (33)$$

where C_w = concentration of PCBs in water in mg/L, and AI = the acceptable intake for prevention of acute and carcinogenic adverse health effects, in mg/day. If fish caught in PCB-contaminated surface water are eaten, and if the same water is the source of drinking water, the allowable concentration of PCBs (C_w mg/day) should be determined as

$$C_w = \frac{AI}{2\ L/day + F \cdot BCF} \qquad (34)$$

where F is the daily fish consumption, BCF is the bioconcentration factor (31,200 L/kg) (U.S. EPA, 1980b; U.S. EPA, undated). The national average of fish consumption is 0.0065 kg/day (U.S. EPA, 1984b). However, it is more appropriate to use regional values where such data are available.

The variabilities of input values needed in Eq. (24) (appropriate for PCB exposure pathways) are wide-ranging for some values, and narrow for others. The inhalation rate of air used for calculation is 20 m^3/day for both adults and children (U.S. EPA, 1985d). Soil ingestion rates used for evaluating short-term exposures are 3 and 0.6 g/day, representing conditions with and without pica, respectively (further explained in Section 15). One lifetime exposure evaluation is based on an average daily rate of 0.6 g/day multiplied by factors to correct for the changing weight of the body as a person grows from a child to an adult. This exposure is assumed to occur from age 1 to 5 years. However, the soil ingestion rate of 3 g/day is also used in long-term exposure evaluation. The absorption factors for PCBs through the gastrointestinal tract for ingested soil, through the pulmonary organs for inhaled air, and through the skin for contacted soil are assumed to be 0.3, 0.5, and 0.05, respectively (U.S. EPA, 1984a; U.S. EPA, 1985e). The off-site factor is assumed to be 1 for longer-term (10-day) exposure evaluations, and 0.5 for lifetime exposure evaluations, using the carcinogenic potency factor. A similar approach can be used for short-term (1-day) and lifetime noncancer exposure evaluations. However, these evaluations are not performed because of a lack of data regarding health effects.

If all intake routes, including drinking water, soil ingestion, air inhalation, dermal contact, and intake of PCBs by means of fish or other food are of relevant importance, the allowable concentration levels can also be combined in similar fashion. Since the scope of the present study pertains to site cleanup, the applicable formulas for combining concentrations are not presented, but they should be considered as the situation warrants.

13. WATER QUALITY LIMITS

The concentration levels of PCBs in drinking water are based on single-route exposures that are estimated to result in acute and chronic toxic effects. This does not imply that bioaccumulation in aquatic organisms does not occur. The assumption pertains to absence of fish contaminated with PCBs in the diet. If other routes are of concern, the allowable concentrations in water should be redefined. The following levels of PCBs in drinking water, corresponding to 10-day AIs, can be calculated for children and adults:

- 10-day health advisory:

$$\text{Safe concentration} = \frac{1\ \text{mg/kg}\cdot\text{day}\cdot 10\ \text{kg}}{100\cdot 1\ \text{L/day}} = 0.1\ \text{mg/L}\ (= 100\ \text{ppb})\ (\text{child})$$

$$\text{Safe concentration} = \frac{1\ \text{mg/kg}\cdot\text{day}\cdot 70\ \text{kg}}{100\cdot 2\ \text{L/day}} = 0.35\ \text{mg/L}\ (= 350\ \text{ppb})\ (\text{adult})$$

Similarly, the concentration levels at the various upper-bound cancer risks assumed are calculated, and the results can be tabulated as follows:

Upper-bound cancer risk	Advisory level (ng/L)
10^{-4}	875
10^{-5}	87.5
10^{-6}	8.75
10^{-7}	0.9

Example chronic toxicity advisory level (at 10^{-6} maximum risk)

$$\frac{10^{-6}\ \text{risk}\ 70\ \text{kg}}{4(\text{mg/kg}\cdot\text{day})^{-1}\cdot 2\ \text{L/day}} = 8.75\text{x}10^{-6}\ \text{mg/L}\ (=8.75\ \text{ng/L})$$

As indicated previously, an Aroclor constitutes a mixture of many congeners. Since each congener compound exhibits different solubility characteristics, the applicability of these limits to individual congeners is ion question. In the absence of short-term data for non-carcinogenic effects, the 10-day health advisory may be used as the 1-day health advisory for commercial Aroclors.

14. LEACHATE CONTAMINATION OF GROUNDWATER

Contaminated leachate will impact groundwater quality. To date, groundwater monitoring data showing major contamination of groundwater by PCBs has been rarely reported. If the contaminated site is located above an unsaturated zone, soil through which leachate has to migrate to reach groundwater will adsorb PCBs and will greatly retard PCB migration, as evidenced by the high soil-water partition coefficients. Experimental work (U.S. EPA, 1980a) has shown that the adsorption characteristics vary depending upon the type of soil used. The experimental values are comparable to the partition coefficients estimated from the values of K_{ow} (water-octanol partition coefficient) given in Table 4. PCBs entering groundwater at hazardous waste sites could also be retarded as they are carried along the flow lines.

The area-source groundwater model (Hwang, 1985) has been used to evaluate the extent of retardation and dilution of contaminants in groundwater. A typical precipitation rate has been used to estimate a leachate generation rate which is a source term in the groundwater rate and transport model. Two different values of the retardation factor covering the extreme variations of the soil-water partition coefficients were considered: $R_d = 127$, corresponding to $K_d = 22\ \text{cm}^3/\text{g}$; and $R_d = 5715$, corresponding to $K_d = 1000\ \text{cm}^3/\text{g}$, where K_d represents the soil-water partition coefficient, and R_d is the retardation factor ($R_d = 1 + \frac{P_b}{e} K_d$, P_b = bulk density, e = porosity). The results of modeling show that when the concentration of PCBs in leachate is maintained at 0.12 mg/L, the vertically averaged PCB concentration in groundwater at 1000 cm away from the center of a disposal site after two years of release is 0.5×10^{-4} mg/L and 1.9×10^{-7} mg/L for the low and high values of the retardation factor, respectively. Other parameter values used in this simulation were: leachate flow rate = 23.4 cm^3/s, groundwater seepage velocity = 5 x 10^{-4} cm/s, porosity of groundwater medium = 0.35, depth of the aquifer = 300 cm, size of disposal site = 0.5 acre, and the bulk density of the medium = 2 g/cm^3. The simulation was repeated for a distance of 1 km away from the site. The concentration values at that distance were very small.

The groundwater transport analysis back-calculated allowable leachate concentrations entering groundwater below a hazardous waste facility, given the maximum allowable concentrations at a compliance point. These calculations do not account for "facilitated transport" via dissolved organics, cosolvents, etc. As indicated previously, the maximum allowable concentrations were based on the allowable daily intakes designed to prevent acute and chronic health effects. For the purposes of simulation, the maximum allowable drinking water concentrations at such distances as 1000 cm and 1 km from the contaminated site can be estimated. For acute toxicity, the drinking water concentration of 0.1 mg/L is assumed; for chronic carcinogenic toxicity, the concentration of PCB in groundwater assumed was 8.7 ng/L, corresponding to a 10^{-6} lifetime risk.

The down-gradient groundwater concentration is a complex function of leachate concentration, dispersion and retardation in groundwater, initial dilution in groundwater, biodegradation (if any), and groundwater velocity. The functional relationship can be found elsewhere (Hwang, 1985), and takes the form

$$C_L = f_g C_w \tag{35}$$

where C_L represents the leachate concentration corresponding to the drinking water concentration C_w at a point of interest, and f_g is a functional relationship which incorporates fate and transport of PCB in the groundwater medium.

At the lower end of the retardation coefficient (R_d = 127), the calculated leachate concentrations under steady-state conditions are 570 mg/L and 4.3 x 10^{-3} mg/L, for acute and chronic levels, respectively, for the concentrations maintained at the distance of 1000 cm from the site.

The solubility values in Table 4 show that the 10-day acute PCB levels in leachate are above the solubility limits. The solubilities for most Aroclors are above the leachate concentration necessary to prevent the chronic effect of PCBs in groundwater. In other words, the chances of exceeding the level of PCBs in groundwater which would cause acute toxicity would be small, while leachate can enter groundwater to exceed chronic concentration levels. It should be noted that the assumptions used in the calculations are that the points of exposure are 1000 cm and that the concentrations attainable under steady-state conditions are used. For different compliance points, the allowable leachate concentrations will change. In addition, steady-state conditions assume that the transport of PCBs in groundwater occurred for a long time, so that concentrations at the exposure point are no longer changing. However, the maximum allowable leachate concentrations for other conditions of transport, and for different locations of exposure, can also be evaluated. For individual congeners, the concentration levels should be reevaluated because of the difference in chemical and physical characteristics between an individual congener and an Aroclor consisting of an array of congener mixtures.

15. SOIL INGESTION PATHWAY

A literature search shows that there is very limited information on the rate of likely soil ingestion by children and adults which can be used in exposure assessment. The situation for which the information is derived differs from study to study. Lepow (1975) studied the mouthing behavior of ten 2- to 6-year-old children in connection with investigations into the principal cause of the excessive lead accumulation in the children. The total soil ingestion rate for a 2-year-old child based on the average amount of street dirt, house dust, and soil ingested by the child by putting his hands and fingers in his mouth, can be summed as 0.6 g of soil per day.

Wedeen et al. (1978) observed the lead concentration in blood of a black woman with a 12-year history of habitual eating of garden soil. Using the levels of blood lead concentration and the concentrations of lead in the soil analyzed, they estimated the amount of lead the subject had consumed each year from her garden soil. From this estimate, the soil ingestion rate is estimated to have been in the range of between 1.96 and 3.9 g/day, with an average value at about 3 g/day. The lead concentration in the dried garden soil is reported to be between 690 μg/g and 700 μg/g of soil.

Investigators at the Centers for Disease Control present the lifetime ingestion rate of contaminated soil according to age group (Kimbrough et al., 1984). The paper states that the data presented are "based on work done studying lead uptake from contaminated soils." However, the writers of this report were unable to locate the original experimental work or its source to cite in this evaluation. The ingestion rate is assumed to change at different ages, and is given as 0 for the age group 0 to 9 months, as 1 g/day for the age group 9 to 18 months, as 10 g/day for the age group 1.5 to 3.5 years, as 1 g/day for the age group 3.5 to 5 years, and as 0.1 g/day for a 5-year-old child.

The second column of Table 8 shows the lifetime carcinogenic risk posed by ingesting soil contaminated with PCBs at various concentrations. This table is prepared using Eq. (6) at the soil ingestion rate of 3 g/day for children aged 1 through 6 and an average weight of 10 kg. The values for other parameters used are SM = 0.5, GI = 0.3, and k = 0. The risk values in the second column compare with those in the third column, which are prepared using the soil ingestion rate applicable to different age groups, as suggested by the Centers for Disease Control.

TABLE 8. MAXIMUM LIFETIME RISK FOR INGESTING SOIL CONTAMINATION AT DIFFERENT PCB LEVELS

	Lifetime risk	
PCB level in soil (μg/g)	(IR = 3)	Age-dependent IR[a]
0.1	1.54×10^{-6}	1.92×10^{-6}
1	1.54×10^{-5}	1.92×10^{-5}
5	7.7×10^{-5}	9.6×10^{-5}
10	1.54×10^{-4}	1.92×10^{-4}
20	3.08×10^{-4}	3.8×10^{-4}
50	7.7×10^{-4}	9.6×10^{-4}

[a] Taken from Kimbrough et al., 1984.

A computer program was convenient to use in carrying out the summation of daily intakes over a lifetime period. The lifetime risk represents an upper-bound estimate of the unit risk that can occur as a result of ingesting PCB-contaminated soil over a lifetime, and indicates the risk posed by a single exposure pathway; that is, soil ingestion is the sole route for PCB intakes, and other pathways, including air, water, fish are assumed to be insignificant sources of human intake of PCBs. Since the population that will be subject to soil ingestion resides in the area and must breathe the air affected by PCB emissions, the magnitude of PCB intakes by the ingestion and inhalation routes needs to be compared to determine the significant pathway. The comparision is presented in Section 18.

Similarly, in order to determine the daily health advisory levels for a single exposure pathway, the daily PCB intakes equivalent to ingesting 3 g of soil in a day at various PCB concentrations are calculated. The results are shown in Table 9.

TABLE 9. MAXIMUM DAILY PCB INTAKE BY INGESTION OF SOIL AT VARIOUS PCB CONCENTRATIONS

PCB level in soil (μg/g)	Daily PCB intake at 30% absorption (mg/day)
0.1	0.00009
1	0.0009
5	0.0045
10	0.009
20	0.018
50	0.045

The exposure pathways for soil ingestion, air inhalation, and other routes must be evaluated. If one pathway is found to be dominant over the other, the insignificant pathway based on short-term and long-term intake rates can be ignored. If they are comparable, the concentration levels need to be adjusted to reflect the combined intake rates by using Eq. (24) or combinations of Eqs. (8) through (22).

16. INHALATION PATHWAY

16.1. INTAKE BY AIR EXPOSURE ROUTE

Exposure to PCBs occurs at or near contaminated sites through inhalation of ambient air contaminated with PCB vapors or particulate matter on which PCBs are adsorbed. The PCB vapors emitted from contaminated soil will be diluted by the action of winds before a person inhales the ambient air. When PCBs are adsorbed on soil, the vapor pressure of the PCBs above the soil surface will be always less than the vapor pressure exerted by PCBs when they are present in pure form. In other words, the adsorption phenomena depress the vapor pressure that can exist under saturated conditions. This true vapor pressure is referred to as "partial pressure." When adsorption reaches its saturation capacity on soil, the partial pressure will be equal to the pure PCB vapor pressure.

Estimates of PCB concentrations in the ambient air impacting the population at hazardous waste sites are discussed in this section, as well as comparison with the intake rates of PCB through soil ingestion. In calculating ambient air PCB concentrations, the first task was to estimate the emission rates of PCBs from the bulk of soil contaminated at various concentrations of PCBs. The emission rate calculations can be rigorously performed by the methods summarized by Hwang (1982) for steady state conditions, and by methods presented in the Appendix for transient conditions.

Based on the inhalation rate of 20 m^3/day, and absorption rates of 50% and 30% for inhaled and ingested PCBs (U.S. EPA, 1984b), respectively, the concentrations of PCBs in inhaled air and particulates equivalent to the dosage causing acute and chronic toxic effects can be estimated. The purpose of this exercise is to evaluate the concentrations of PCBs in the air, which are comparable to the ingestion dosage. Tables 10 and 11 show this comparison for intake rates corresponding to acute and chronic effects respectively.

Table 10, for example, shows that a daily intake of 3 g of soil containing 5 μg/g of PCBs is equivalent to a daily inhalation of air containing 0.45 $\mu g/m^3$. The concentration of PCBs on particulate matter must be as high as 7,500 μg/g for the inhalation of particulates at an assumed concentration of 60 $\mu g/m^3$ to be comparable to the ingestion of soil and inhalation of air described above. This concentration is used because the concentration should not exceed the primary ambient air quality of 75 $\mu g/m^3$ for particulate matter. Since the concentrations of PCBs on soil under consideration are in the range which is less than this concentration, it can be assumed that the PCB intake by inhalation of particulate matter at contaminated sites is relatively unimportant. Similar arguments can be made for the results shown in Table 11 for long-term intakes. The equivalent air concentrations C_e shown in Tables 10 and 11 are calculated by the following formula:

$$C_e\ (\mu g/m^3) = \frac{\text{daily intake (mg/day)} \times 10^3\ \mu g/mg}{20\ m^3/\text{day}\ 0.5\ \text{(absorption factor)}} \qquad (36)$$

16.2. EMISSION EVALUATION SCENARIOS

Emission rates are estimated for four different scenarios: Case 1--as the PCBs volatilize from the initial contaminated soil column, they are depleted from the column of soil by diffusional transfer of PCBs across the soil-air interface, resulting in unsteady-state emission rates, and the layer depleted of PCBs acts as cover material retarding the volatilization rate; Case 2--the same scenario as in Case 1 except that the contaminated soil is initially covered with 25 cm of cover material; Case 3--PCBs are volatilized from the

TABLE 10. COMPARISON OF PCB INTAKES BY INGESTION AND INHALATION ROUTES FOR ACUTE EFFECTS

PCB levels in soil (ppm)	Daily acute intake (mg/day)	Equiv. air conc. for acute ingestion (ug/m^3)	Conc. of PCBs on particulates (µg/g)
0.1	0.00009	0.009	150
1	0.0009	0.09	1,500
5	0.0045	0.45	7,500
10	0.009	0.9	15,000
20	0.0018	1.8	30,000
50	0.045	4.5	75,000

TABLE 11. COMPARISON OF PCB INTAKES BY INGESTION AND INHALATION ROUTES FOR CARCINOGENIC EFFECTS

PCB levels in soil (μg/g)	Lifetime risk at IR = 3	Average daily intake (mg/day)	Equiv. air conc. for the risk (μg/m^3)	Conc. of PCBs on particulates (μg/g)
0.1	1.54×10^{-6}	3.86×10^{-6}	0.00039	6.4
1	1.54×10^{-5}	3.86×10^{-5}	0.0039	64.3
5	7.7×10^{-5}	1.93×10^{-4}	0.019	322
10	1.54×10^{-4}	3.86×10^{-4}	0.039	643.7
20	3.08×10^{-4}	7.72×10^{-4}	0.077	1287
50	7.7×10^{-4}	1.93×10^{-3}	0.19	3217

surface of contaminated soil, and the PCB concentration at the surface is kept at a constant value; Case 4--the contaminated soil is covered with 25 cm of clean cover soil to retard the volatilization rate, and the concentration of PCBs at the surface is kept at a constant value.

As pointed out previously, there exists a PCB saturation point above which the air-phase PCB concentration in equilibrium with (or partitioned with) the contaminated soil remains constant, and hence the emission rate of PCBs will also remain essentially steady over time. Below this point, a concentration profile of PCBs across the contaminated soil column starting from the surface to the depth of contamination will be created as volatilization progresses. This will result in unsteady-state emission rates which will vary over the period that exposure occurs. The period considered includes 10 days for 10-day advisory, and estimated lifetime (70 years) for long-term advisory.

The concentration of PCBs in soil corresponding to the saturation point can be estimated from the knowledge of vapor pressure and air-soil partitioning. For example, since the vapor pressure of Aroclor 1254 (7.71×10^{-5} mmHg) as reported in a publication, corresponds to the saturation concentration of 1,362.7 $\mu g/m^3$, the PCB concentration in soil at the saturation point is

$$C_s = \frac{1{,}362.7\ \mu g/m^3}{[K_{as}(g\ soil/cm^3\ air) \times 10^6\ cm^3/m^3]}$$

$$= \frac{1{,}362.7\ (2.44 \times 10^{-2})(1{,}000)}{(8.37 \times 10^{-3} \times 10^6)} \cong 4\ \mu g/g$$

for an assumed value for K_d of 1,000 cm^3/g. The saturation concentration is dependent upon the value of the air-soil partition coefficient. PCB saturation concentrations in air, and the corresponding concentrations in soil for the Aroclors considered as part of this evaluation, are tabulated in Table 12, based on the soil-water partition coefficient of 1,000 cm^3/g for highly adsorbable earth material (U.S. EPA, 1980a), which is used in calculating the air-soil partition coefficient. A similar table can be prepared at the lower end value of the soil-water partition coefficient, which is approximately 40 cm^3/g for sandy material (U.S. EPA, 1980a).

Case 3 and perhaps Case 4 may be unrealistic, because as volatilization continues, the PCB concentration in soil decreases and the surface layer, depleted of PCBs, should act as an uncontaminated layer decreasing the emission rate. But this estimate should provide upper-bound values for emission rates. Cases 3 and 4 would be applicable in real-case situations when the concentration of PCBs in soil is high enough so that the air-phase concentration in equilibrium with soil remains constant until the concentration of PCBs in soil decreases to the saturation point (C_{sm}, as defined on p. 12-5). Below the saturation point, the air-phase concentration will no longer remain constant, but will decrease in direct proportion to the soil-phase concentration. In the emission rate calculation, the partial pressure of PCBs as a result of partitioning between the air and soil phases is used. Since the vapor pressures and Henry's Law constants are different for most PCBs, some typical PCBs are used for the purpose of illustrative calculations. Table 13 summarizes the results of calculations for emission rates for soil containing 1 μg of Aroclor-1254 and Aroclor-1242 per g of soil. The values shown for Cases 1 and 2 are the averages for one day emitted after the initial contamination at the concentration. This is for illustration only because Aroclor-1248 and Aroclor-1260 are also used for emission rate calcualations. The models used for the emission rate estimation and necessary calculations are shown in Appendix A. The emis-

TABLE 12. CONCENTRATION OF PCBS IN SOIL AT SATURATION VAPOR PRESSURE BASED ON K_d = 1000 cm^3/g

	PCB concentration in soil with saturated vapor (μg/g)	Saturated vapor concentration (μg/m^3)
Aroclor-1254	4	1362.7
Aroclor-1242	250	5823.3
Aroclor-1260	28.2	822.8
Aroclor-1248	55.3	7962.8

TABLE 13. PCB EMISSION RATES FROM 1 μg/g PCB SOIL AT DIFFERENT CONTROL LEVELS

	Emission rates (g/cm^2·s)	
Scenario	Aroclor-1254	Aroclor-1242
Case 1	1.03×10^{-11}	2.7×10^{-12}
Case 2	3.67×10^{-13}	2.8×10^{-14}
Case 3	1.13×10^{-10}	8.57×10^{-12}
Case 4	1.67×10^{-13}[a]	1.14×10^{-14}[a]

[a] The models for estimating emissions from landfills underpredict the emission rate in comparison to Case 2. The estimates for Cases 2 and 4 are based on the mathematical model (described in Appendix A) and the empirical model (Farmer et al., 1980), respectively.

sion rates at various concentration levels of PCB in soil are made for evaluating the impact of volatilization on the exposed population at various locations. Table 13 shows that the emission rate of Aroclor-1254 is different from that of Aroclor-1242 at the same soil contamination level. The table also shows that the use of cover material is very effective in reducing the emission rate. The average emission rates over a period of 10 days, or a lifetime, for Cases 1 and 2 can be similarly estimated by the rigorous mathematical formulae provided in Appendix A. As PCBs volatilize, the partial pressure of PCBs at the soil-air interface decreases, and the soil layer, depleted of PCBs, provides the barrier for mass transfer, causing the emission rates given for Cases 3 and 4 to approach values comparable to those for Cases 1 and 2, respectively. The Thibodeaux and Hwang model (1982), originally developed for land treatment facilities, provides emission rates similar to those shown for Case 1 and 2 in Table 13.

PCBs volatilized into the atmosphere from a contamination site will impact the population in the surrounding region. The concentrations of PCBs at the point of impact need to be determined in order to evaluate the significance of the air emissions compared with the soil ingestion and dermal pathways. Acute and chronic impacts are based on the daily concentrations and the concentrations averaged over an annual period. Emission rates corresponding to all four cases of maintenance levels are used to estimate the concentrations of PCBs in the ambient air at the site and at distances of 0.1 km and 1 km.

16.3. AIR DISPERSION MODELING

Dispersion modeling is used to estimate the ambient air concentrations which may be possible for daily and annual exposures. Dispersion modeling for estimating the annually averaged concentrations makes use of six stability classes, six wind speed classes, and 16 sectors, assigning the receptor point to one of the 16 sectors. The wind rose data consisting of 6 x 6 x 16 = 576 elements are compiled by the National Climatic Center in Asheville, North Carolina. The dispersion model for the annual concentration sums the concentration contributions over the entire range of stability classes and wind speeds for each exposure location downwind of the contamination site, and can take the following form (Bruce, 1969):

$$C(X,k) = 2.03 \times 10^{6} Q \sum_{i=1}^{6} \frac{1}{(\alpha_z)_{i,X}} \sum_{j=1}^{6} \frac{f_{ijk}}{U_j} \qquad (37)$$

where C(X,k), the annual concentration located in a sector k at a distance of X downwind of the site ($\mu g/m^3$);$(\alpha)i,X$ = standard deviation of the plume in the z-direction (vertical direction) at distance X for stability class i, Q = emission rate, g/s, U_j = mean wind speed for wind speed class j, m/s and f_{ijk} = frequency of wind in stability class i, wind speed class j, and direction in sector k. Both X and the standard deviation have the the units of meters.

The values for the standard deviation can be found in an air pollution textbook (Wark and Warner, 1981), or can be determined by a curve-fitting equation of the form

$$(\alpha_z)_{i,X} = aX^{b} + d \qquad (38)$$

where a, b, and d are empirical constants varying according to stability i and distance X (Wark and Warner, 1981; Martin, 1976). The values for these constants are given in Table 14 (Martin, 1976).

TABLE 14. VALUES OF CONSTANTS FOR STANDARD DEVIATION EXPRESSION AS A FUNCTION OF DOWNWIND DISTANCE AND STABILITY CONDITION

	X < 1 km			X > 1 km		
Stability	a	b	d	a	b	d
A	440.8	1.941	9.27	459.7	2.094	-9.6
B	106.6	1.149	3.3	108.2	1.098	2.0
C	61.0	0.911	0	61.0	0.911	0
D	33.2	0.725	-1.7	44.5	0.516	-13.0
E	22.8	0.678	-1.3	55.4	0.305	-34.0
F	14.35	0.740	-0.35	62.6	0.180	-48.6

The estimation of the on-site ambient air concentration does not require the use of air dispersion modeling presented above. The ambient air concentration is controlled by the extent of dilution before dispersion occurs downwind of the source. The dilution can be estimated from the knowledge on the rate of PCB emissions and volumetric rate of wind being mixed with PCB vapors. The method for estimating the on-site ambient air concentrations is described in detail in Appendix A.

16.4. AIR EXPOSURE EVALUATION

Table 15 summarizes the results of dilution estimation and dispersion modeling to obtain the concentration levels of PCBs in ambient air at various locations considered for emissions of PCB-1254. This table is a summary of one set of calculations for the PCB concentration of 1 µg/g in soil for each scenario. The wind speed of 10 mph is used for both one-day and annual concentration averages. The Climatic Atlas of the United States provides information on annual average wind speed. A default value of 10 mph represents a typical annual wind speed in the United States. A site-specific evaluation will require detailed wind rose information based on local measurements.

TABLE 15. AMBIENT PCB CONCENTRATIONS AT DIFFERENT LOCATIONS (PCB IN SOIL = 1 μg/g, PCB-1254)

	Concentrations (μg/m^3)				
		0.1 km from site		1 km from site	
	On-site	Daily	Annual	Daily	Annual
Case 1	0.61	0.026	0.0065	0.0016	0.0004
Case 2	1.4×10^{-3}	5.9×10^{-5}	1.47×10^{-5}	3.5×10^{-6}	8.9×10^{-7}
Case 3	11	0.48	0.12	0.03	7.3×10^{-3}
Case 4	0.017	7.1×10^{-4}	1.8×10^{-4}	4.3×10^{-5}	1.1×10^{-5}

The standard deviation curve for D stability is employed in estimating the ambient air concentrations at the distances of 0.1 and 1 km from the site as shown in Table 15, since D stability is by far the most frequently occurring stability class. Although D is the most common stability, an exposure-weighted average stability should be used for site-specific evaluations. The frequency with which winds blow toward a sector of interest is assumed to be 1 for evaluating the worst-case daily concentrations, while it should be based on the most common of the standard 16 wind directions for estimation of the average annual concentration levels. The concentrations in ambient air on-site and at distances of 0.1 km and 1 km from the site are given in the table. Calculations are performed for the ambient air concentrations of PCB-1242, PCB-1248, and

PCB-1260, but these are not tabulated here. The values in the table should not be construed as representing fixed ambient air concentrations at the location and under the mode of exposure. The values are presented by way of illustration to compare the contributions to ambient air occurring given the different assumed conditions.

The concentration of PCBs in equilibrium with soil containing 1 μg/g PCB-1254, which corresponds to the partial pressure of PCBs partitioned above the soil, is 340 $\mu g/m^3$. This represents the maximum vapor concentration when PCB is emitted from the soil surface. Based on this concentration and the estimated ambient air concentrations given in Table 15, one can calculate the dilution factors of the emissions for use in Eq. (9) or (24). For example, the air dilution factor for the on-site exposure corresponding to the Case 1 emission

rate would be

$$D = 0.611/340 = 0.0018$$

The values shown in Table 15 can also be used to determine the daily intakes and lifetime risk levels corresponding to breathing each ambient air level. For example, the daily intake from the exposure to the ambient air concentrations of PCB-1254 at 0.1 km from the site for the Case 1 emission rate can be based on a daily inhalation rate of 20 m^3/day of air and 50% absorption factor for inhalation.

$$\text{Daily intake (mg/day)} = C(\mu g/m^3) \cdot 20\ m^3/\text{day} \cdot (1/1000\ mg/\mu g)$$

$$= 0.026\ (20)(1/1000)(0.5) = 0.00026\ \text{mg/day}$$

The same daily intake can be obtained by using Eq. (9):

$$\text{Daily intake} = K_{as} \cdot C_s \cdot D \cdot IH \cdot 10^{+3}(ABA)(SM)$$

$$= (8.37\text{x}10^{-3}/1000)(1/2.44\text{x}10^{-2})(1)(0.026/340)(20)(10^3)(0.5)(1)$$

$$= 0.00026 \text{ mg/day}$$

Similarly, the lifetime risk associated with breathing the ambient air can be calculated as follows:

$$\text{Risk} = C(\mu g/m^3) \cdot 20\ m^3/\text{day} \cdot 1/70\ \text{kg} \cdot 1/1000\ \text{mg}/\mu g\ (4)\ (\text{mg/kg/day})^{-1}\ (0.5)(0.5)$$

where C is the ambient air concentrations shown in Table 15 and the value 4 $(\text{mg/kg} \cdot \text{day})^{-1}$ represents the potency factor for PCBs, and an additional factor of 0.5 is the off-site factor under the assumption that a resident stays in the area 50% of the time.

A series of calculations can be performed as shown above, or the procedure shown above can be reversed to back-calculate the PCB contaminations in soil which will provide the allowable ambient air concentrations at the locations considered and at the acute and chronic effect levels.

17. DERMAL CONTACT PATHWAY

Deposition of contaminated soil, dirt, or dust on human skin can provide another pathway for human intake of PCBs. PCBs can be absorbed through skin when PCB-contaminated particulates come into contact with skin. Exposure evaluation requires an estimation of the amount of the particulates on skin, and the extent or rate of absorption. The absorption rate is dependent upon the type of chemicals. Some chemicals are readily absorbed, while others are not.

There are many factors affecting the amount of soil which can be accumulated on skin. Factors include the exposed human skin area, contact time, type of soil, soil conditions, and type of activities. For example, the amount deposited on children playing in a contaminated area may be different from that on adults working in a garden.

OHEA (U.S. EPA, 1984b) has made an estimation of the amount of soil deposition on skin based on the studies by Lepow (1975) and Roels et al. (1980). Both investigators, using adhesive tape, measured the amount of soil and dirt accumulated by children on exposed areas such as hands, palm, and fingers. The measured amount of soil ranges from 0.5 to 1.5 mg/cm^2, with an average value of 1 mg/cm^2. It should be noted that this is an average value over the surface of the exposure area, and that some parts of the body may have more accumulation of soil than others.

The area of human skin that will come in contact with soil or dirt depends upon the protective measures used during the time that such contact occurs, as well as the age group involved. The exposed surface area of an adult is estimated to range from about 900 to 2,900 cm^2. The exposed surface area of a child may be smaller in proportion to their total surface area.

The ranges of values associated with the factors mentioned above makes it difficult to arrive at an average value for the amount of soil and dirt accumulated on soil. An assumption of an average soil deposition at 1 mg/cm^2, and exposed surface area of about 1,000 cm^2 on a daily basis, provides an average daily deposition rate of 1 g per day. The variability is such that this value may be different by a factor of as much as two.

Investigators at the Centers for Disease Control present an estimated daily deposition of soil on skin according to age group (Kimbrough et al., 1984). Their tabular presentation shows that the daily amount of soil deposited on skin is 0 for the age group 0 to 9 months; 1 g for the age group 9 to 18 months; 10 g for the age group 1.5 to 3.5 years; 1 g for the age group 3.5 to 15 years; and 100 mg at age 15 years.

18. COMPARISON OF EXPOSURES BY SOIL INGESTION, INHALATION, AND DERMAL CONTACT

Table 16 shows comparisons of PCB intake by various exposure routes. Calculations apply for PCB-1254 because the emission rate is dependent upon the soil-air partition coefficient, which is different for each PCB. The ambient air concentration is the on-site value based on the emission rate averaged over 1 day after soil is contaminated up to the surface without cover.

TABLE 16. COMPARISON OF INTAKES BY VARIOUS EXPOSURE ROUTES[a]

Exposure route	Contact rate	Absorption fraction	Daily intake (mg/day)	Lifetime intake (mg/70 yrs)
Soil ingestion				
	3 g/day (with pica)	0.3	9×10^{-4}	1[d]
	0.6 g/day	0.3	1.8×10^{-4}	0.2
Inhalation	$20\ m^3$/day[b]	0.5	6.1×10^{-3}	78[e]
Dermal absorption	1 g/day	0.05	5×10^{-5}	0.64[f]
Dust inhalation	$20\ m^3$/day[c]	0.5	6×10^{-7}	7.7×10^{-3}[e]

[a] Used for illustration: PCB-1254 at concentration of 1 μg/g in soil.
[b] On-site ambient air concentration based on 1-day average emission rate after surface contamination (no cover).
[c] Concentration of suspended particular matter: 60 $\mu g/m^3$.
[d] 183 days/year for 6 years.
[e] Off-site factor = 0.5.
[f] An average exposure of 1.5 mg/cm^2 and the exposure surface area of 1000 cm^2 assumed; off-site factor = 0.5.

Evaluation under the scenario of the use of cover, or longer-term emission averages, may change the daily intake by inhalation considerably. For example, a calculation shows that the use of 25-cm clean soil cover will reduce the daily intake by inhalation to 1.4×10^{-5} mg/day instead of 6.1×10^{-3} mg/day. On the other hand, exposures by soil ingestion, dermal absorption, and dust inhalation will be likely to decrease because clean soil is used. The ingested soil or the soil on the surface of the cover that may be accumulated on skin is initially clean. Hence, the daily intakes by pathways other than inhalation also become small. However, the concentration of PCBs in the initially clean cover material could increase as the PCBs in the air phase being emitted are adsorbed on the cover material as the liquid PCBs rise toward the surface due to capillary potential. The table suggests that soil ingestion and inhalation are two competing exposure routes for PCB intake. The dermal contact can also become a contributing route for some conditions of exposure duration. When a high concentration of particulate matter in the ambient air is prevailing, the comparison shown in Table 16 can no longer apply. Consequently, the contribution to PCB intakes by inhalation of particulate matter will increase. There are a range of other possibilities which may result in a comparison different from that shown in Table 16.

19. RESULTS

19.1. DERIVATION OF PERMISSIBLE SOIL CONTAMINATION

Determination of the permissible PCB levels in soil for intake through the combined exposure routes makes use of 1) Eq. (24) when the soil concentration is below the saturation point; and 2) the summation of Eqs. (8), (9), and (10), equated to the acceptable intake otherwise. For each Aroclor under consideration, a separate exposure evaluation can be made for the following classes of exposure location and route: 1) Exposure occurs on-site. This can be further subdivided into: (a) sites which are readily accessible to children, and hence for which soil ingestion is a possibility, and (b) sites for which there is no possibility of soil ingestion, and hence exposure is only through inhalation; 2) sites which no population is assumed to enter within the radius of 0.1 km from the site; and 3) sites which no population is assumed to enter within the radius of 1 km from the site.

Two classes of soil ingestion rates are evaluated when exposure occurs on-site (Class 1 above). For the first class, estimates of exposure are calculated for a person with pica who consumes 3 g per day between the ages of 1-5 years. For the second class, estimates of exposure are calculated for soil ingestion of 0.6 g per day between the ages of 1-5 years. For both classes, frequency of exposure is assumed to be every other day for lifetime exposures. For a 10-day exposure, soil consumption is assumed to occur consecutively for 10 days. No soil ingestion is assumed for sites which are not accessible to population within 0.1 km or 1 km from the contaminated site. The route of exposure in these cases is by inhalation only. For lifetime inhalation exposure estimates, it is assumed that the population is exposed 50% of the time, i.e., 12 hours/day, or 6 months/year.

The emission rate of volatilized PCBs can be considerably reduced by covering the contaminated soil by low-porosity uncontaminated soil or clay material. The reduction in the emission rate will result in a decrease in ambient air concentrations of PCBs by the action of blowing winds. When PCB material is directly exposed to the atmosphere, the PCB levels in soil required to maintain the same level of exposure will be less than those expected when the PCB material is covered with low-permeability material of appropriate thickness. The cover would also serve as a deterrent to soil ingestion and direct dermal contact.

The worst-case emissions would occur when the contaminated soil is initially exposed to the atmosphere and the soil is contaminated up to the conditions exhibiting saturation vapor pressure. Models are used to estimate emission rates which can be constant or time-varying depending upon the degree of soil contamination. The constant emission rate can be assumed if the vapor phase concentration maintains its constant value at the surface of contamination. There will be a profile along the layer of soil contamination for time-varying emission rates. The models for constant and time-varying emission rates are applied with or without cover material. Calculations corresponding to Cases 1, 2, and 3 for exposure possibilities are repeated at an assumed 25-cm (10-inch) thickness of a soil cover initially free of PCB contamination.

The ambient air concentrations given in Table 15, and the resulting air dilution factors calculated, are based on an annual average wind speed of 10 mph. When wind speed is lower than this, it is possible that the daily dilution factors could be higher than the values in the calculations (less dilution). The combined soil concentration values based on Eq. (24) will be lower when the dilution factor is higher. More accurate considerations of meteorological conditions will require site-specific evaluation.

Among many factors affecting the emission rate (including vapor pressure, soil-air partition coefficient, and Henry's law constant), the variability associated with the soil-air partition coefficient is more pronounced than any other chemical and physical properties. This is caused by the wide variation in experimental values for the soil-water partition coefficient reported in the literature (U.S. EPA, 1980a), ranging from 22 to 2,000 cm^3 water/g soil. For clay and sandy materials, the range includes about 40 to 1,000. The values of K_d higher than 1,000 are obtained from experiments using coal chars.

For each Aroclor considered, the evaluation is performed for each set of conditions. The combination of these conditions can be summarized as follows:

(1) No cover is used. This is designated as the case of "Uncovered Surface Contamination." Contaminated soil surface is left uncovered after removal action in this case.

(2) Soil cover of 25-cm thickness initially free of PCB contamination is used. This is designated as the case of "25-cm-Thick Clean Soil Cover". Contaminated soil surface is covered with 25-cm-thick soil cover. No soaking by liquid PCBs is assumed, which will reduce the effective cover thickness.

(3) On-site exposure evaluation is conducted at two different soil ingestion rates: 0.6 and 3 g/day. The higher rate is reported for an individual with the habit of eating soil (pica). For life exposures, soil is consumed every day for ages 1-5 years. For 10-day exposures, soil is consumed every day for ages 1-5 years.

(4) Inhalation rate is 20 m^3/day for both an adult and a child. Population is exposed for 50% of the time for lifetime exposure durations. Ten-day evaluations are based on a daily inhalation rate of 20 m^3/day for 10 consecutive days.

(5) Noncancer and cancer effects are evaluated at the acceptable intakes corresponding to 10-day exposure on the first day of cleanup and after 10 days of elapsed time upon cleanup, and lifetime permissible exposures.

(6) Two extreme values of the soil-air partition coefficients are used in the evaluation. The high values of K_d (soil-water) correspond to low values of K_{as} (soil-air).

(7) Area contaminated is 45 m x 45 m or approximately 0.5 acres.

The combinations of these evaluation conditions are presented in tabular form in Table 17. The soil ingestion rates of both 3 and 0.6 g/day are used in the evaluation pertaining to the longer-term (≡10-day) intakes. The soil ingestion rate of 0.6 g/day between ages 1 and 5 is used for lifetime exposure evaluation, and this value is averaged with respect to soil ingestion and changing body weight over a lifetime. For each Aroclor, there are 120 different situations demanding different permissible levels of PCBs in soil depending upon the location, route and duration of exposure, elapsed time after site cleanup, and the type of health effects to be protected. Table 18 shows the corresponding values for K_d (soil-water partition coefficient) and K_{as} (soil-air partition coefficient) for Aroclors considered. The high values of K_d correspond to the low values of K_{as}, and vice versa.

19.2. SUMMARY OF RESULTS

The method for determining the permissible PCB levels in soil is programmed in a personal computer to avoid hand calculations. Tables 19 through 22 summarize the results of computerized calculations for the case of "Uncovered Surface Contamination," and Tables 23 through 26 are for the case of "25-cm (10-inch)-Thick Clean Soil Cover." The ranges are presented because the low and high values for the soil-air partition coefficient are used in the evaluation.

TABLE 17. EVALUATION CONDITIONS FOR EACH AROCLOR

Location and route of exposure Intake rates		Values for partition coefficient (K_d cm^3/g) and cover depth (L cm)									
		On-site						0.1 km from site		1 km from site	
		Soil ingestion (3 g/d) inhalation dermal		Soil ingestion (0.6 g/d) inhalation dermal		Inhalation only		Inhalation		Inhalation	
Ten-day intake, child (100 µg/d)	K_d[a]	1,000	40	1,000	40	1,000	40	1,000	40	1,000	40
	L	0[b]	25[c]	0	25	0	25	0	25	0	25
Ten-day intake, adult (700 µg/d)	K_d	1,000	40	1000	40	1,000	40	1,000	40	1,000	40
	L	0	25	0	25	0	25	0	25	0	25
10^{-7} risk (0.00175 µg/d)	K_d	1,000	40	1,000	40	1,000	40	1,000	40	1,000	40
	L	0	25	0	25	0	25	0	25	0	25
10^{-6} risk (0.0175 µg/d)	K_d	1,000	40	1,000	40	1,000	40	1,000	40	1,000	40
	L	0	25	0	25	0	25	0	25	0	25
10^{-5} risk (0.175 µg/d)	K_d	1,000	40	1,000	40	1,000	40	1,000	40	1,000	40
	L	0	25	0	25	0	25	0	25	0	25
10^{-4} risk (1.75 µg/d)	K_d	1,000	40	1,000	40	1,000	40	1,000	40	1,000	40
	L	0	25	0	25	0	25	0	25	0	25

[a] K_d = soil-water partition coefficient in units of cm^3 water/g soil (= conc. in soil/conc. in water), high values close to clays, low values close to sand.

[b] Means no cover designated as "surface contamination."

[c] Means 25 cm (10") clean soil cover applied immediately after remedial action.

TABLE 18. LOW AND HIGH VALUES OF AIR-SOIL PARTITION COEFFICIENT USED IN THE EVALUATION

PCB type	K_d (cm^3 water/g soil)[a] High	Low	K_{as} (g soil/cm^3 air)[b] Low	High
1242	1,000	40	2.35×10^{-5}	5.87×10^{-4}
1248	1,000	40	1.44×10^{-4}	3.6×10^{-3}
1254	1,000	40	3.43×10^{-4}	8.58×10^{-3}
1260	1,000	40	2.92×10^{-4}	7.31×10^{-3}

[a] K_d is the soil-water partition coefficient and has the unit of cm^3 water/g soil which is equivalent to concentration in soil/concentration in water.

[b] K_{as} is the soil-air partition coefficient and has the unit of g soil/cm^3 air, which is equivalent to concentration in air/concentration in soil. This is calculated by (H/K_d) $(1/2.44 \times 10^{-2})$.

TABLE 19. PERMISSIBLE PCB-1242 SOIL CONTAMINATION LEVELS (UNCOVERED SURFACE CONTAMINATION)

Location and route of human exposure	Permissible levels (μg/g) corresponding to					
	Noncancer short-term[a] acceptable intake μg/day[b]		Cancer risk specific doses (μg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	55-60[f]	510-690	0.008-0.01	0.08-0.1	0.8-1.0	8-13
- Soil ingestion[d], inhalation[e]	92-247	2100-2800	0.03-0.06	0.3-0.6	3.0-6.0	35-61
- Inhalation only[e]	116-vs[g]	vs	0.04-0.2	0.4-2.0	4.0-20	110-200
0.1 km from contaminated site - Inhalation only[e]	vs	vs	4.0-20	110-200	1.1x10^4	vs
1 km from contaminated site - Inhalation only[e]	vs[g]	vs	310-430	3.1x10^4	vs	vs

[a] Short-term ≅ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] Ranges result in each case because 1) four PCBs (1242, 1248, 1254, 1260) are considered, each with a different vapor pressure, and 2) high and low values for soil-air partition coefficient are used in the calculations.

[g] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquids for the limit.

TABLE 20. PERMISSIBLE PCB-1248 SOIL CONTAMINATION LEVELS (UNCOVERED SURFACE CONTAMINATION)

Location and route of human exposure	Permissible levels (μg/g) corresponding to					
	Noncancer short-term[a] acceptable intake μg/day[b]		Cancer risk specific doses (μg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	32-80[f]	612-710	0.01	0.1	1.0	8-10
- Soil ingestion[d], inhalation[e]	42-330	2500-2900	0.02-0.04	0.2-0.5	2.0-5.0	37-49
- Inhalation only[e]	47-vs[g]	vs	0.02-0.08	0.2-0.8	2.0-8.0	87-110
0.1 km from contaminated site - Inhalation only[e]	vs	vs	2.0-8.0	90-110	8.7×10^3	8.7×10^5-vs
1 km from contaminated site - Inhalation only[e]	vs	vs	250-270	2.4×10^4-2.5×10^4	vs	vs

[a] Short-term ≅ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] Ranges result in each case because 1) four PCBs (1242, 1248, 1254, 1260) are considered, each with a different vapor pressure, and 2) high and low values for soil-air partition coefficient are used in the calculations.

[g] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquids for the limit.

TABLE 21. PERMISSIBLE PCB-1254 SOIL CONTAMINATION LEVELS (UNCOVERED SURFACE CONTAMINATION)

Location and route of human exposure	Permissible levels (µg/g) corresponding to					
	Noncancer short-term[a] acceptable intake µg/day[b]		Cancer risk specific doses (µg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	90-100[f]	720-730	0.009-0.01	0.09-0.1	1.0-2.0	12
- Soil ingestion[d], inhalation[e]	370-420	2980-3000	0.01-0.04	0.1-0.4	3.0-4.0	36-59
- Inhalation only[e]	vs[g]	vs	0.01-0.05	0.1-0.5	5.0-7.0	460-470
0.1 km from contaminated site - Inhalation only[e]	vs	vs	5.0-7.0	460-470	4.7×10^4	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	1.3×10^3	1.3×10^5	vs	vs

[a] Short-term ≡ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] Ranges result in each case because 1) four PCBs (1242, 1248, 1254, 1260) are considered, each with a different vapor pressure, and 2) high and low values for soil-air partition coefficient are used in the calculations.

[g] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquids for the limit.

TABLE 22. PERMISSIBLE PCB-1260 SOIL CONTAMINATION LEVELS (UNCOVERED SURFACE CONTAMINATION)

Location and route of human exposure	Permissible levels (μg/g) corresponding to					
	Noncancer short-term[a] acceptable intake μg/day[b]		Cancer risk specific doses (μg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	25-87[f]	640-710	0.01	0.1	1.0	12-17
- Soil ingestion[d], inhalation[e]	61-360	2670-2900	0.01-0.04	0.1-0.4	1.0-4.0	40-48
- Inhalation only[e]	vs[g]	vs	0.01-0.06	0.1-0.6	1.0-6.0	77-91
0.1 km from contaminated site - Inhalation only[e]	vs	vs	6-220	90-2.2×10^4	7.7×10^3-vs	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	220-240	2.2×10^4	vs	vs

[a] Short-term ≡ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] Ranges result in each case because 1) four PCBs (1242, 1248, 1254, 1260) are considered, each with a different vapor pressure, and 2) high and low values for soil-air partition coefficient are used in the calculations.

[g] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquids for the limit.

TABLE 23. PERMISSIBLE PCB-1242 SOIL CONTAMINATION LEVELS
(25-cm-THICK CLEAN SOIL COVER)

Location and route of human exposure	Permissible levels (µg/g) corresponding to					
	Noncancer short-term[a] acceptable intake µg/day[b]		Cancer risk specific doses (µg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	170-200[f]	1200-1400	0.03-0.2	0.3-2.0	3-17	170-vs
- Soil ingestion[d], inhalation[e]	450-820	3100-5700	0.1-0.6	1.0-6.0	12-48	260-vs
- Inhalation only[e]	vs[g]	vs	0.9-1.0	9-vs	86-vs	vs
0.1 km from contaminated site - Inhalation only[e]	vs	vs	85-vs	vs	vs	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	vs	vs	vs	vs

[a] Short-term ≡ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] Ranges result in each case because 1) four PCBs (1242, 1248, 1254, 1260) are considered, each with a different vapor pressure, and 2) high and low values for soil-air partition coefficient are used in the calculations.

[g] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquids for the limit.

TABLE 24. PERMISSIBLE PCB-1248 SOIL CONTAMINATION LEVELS
(25-cm-THICK CLEAN SOIL COVER)

Location and route of human exposure	Permissible levels (μg/g) corresponding to					
	Noncancer short-term[a] acceptable intake μg/day[b]		Cancer risk specific doses (μg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	160-190[f]	1100-1300	0.01-.09	0.1-1.0	1-10	26-460
- Soil ingestion[d], inhalation[e]	650-vs[g]	4500-vs	0.02-0.1	0.2-1	2.0-10	93-2,500
- Inhalation only[e]	vs	vs	0.02-0.1	0.2-1	2.0-14	1.9×10^4
0.1 km from contaminated site - Inhalation only[e]	vs	vs	2-14	1.9×10^4	vs	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	vs	vs	vs	vs

[a] Short-term ≡ 10-day intake.
[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.
[c] Children ages 1-5, with pica (consuming 3 g soil/day).
[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).
[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.
[f] Ranges result in each case because 1) four PCBs (1242, 1248, 1254, 1260) are considered, each with a different vapor pressure, and 2) high and low values for soil-air partition coefficient are used in the calculations.
[g] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquids for the limit.

TABLE 25. PERMISSIBLE PCB-1254 SOIL CONTAMINATION LEVELS
(25-cm-THICK CLEAN SOIL COVER)

Location and route of human exposure	Permissible levels (μg/g) corresponding to					
	Noncancer short-term[a] acceptable intake μg/day[b]		Cancer risk specific doses (μg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	140-180[f]	970-1300	0.01-0.09[f]	0.1-0.2	2.0-7.0	vs
- Soil ingestion[d], inhalation[e]	520-vs[g]	4000-vs	0.02-0.06	0.2-0.6	8.0-9.0	vs
- Inhalation only[e]	vs	vs	0.02-0.08	0.2-0.8	10-14	vs
0.1 km from contaminated site - Inhalation only[e]	vs	vs	10-14	vs	vs	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	vs	vs	vs	vs

[a] Short-term ≅ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] Ranges result in each case because 1) four PCBs (1242, 1248, 1254, 1260) are considered, each with a different vapor pressure, and 2) high and low values for soil-air partition coefficient are used in the calculations.

[g] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquids for the limit.

TABLE 26. PERMISSIBLE PCB-1260 SOIL CONTAMINATION LEVELS (25-cm-THICK CLEAN SOIL COVER)

Location and route of human exposure	Permissible levels (μg/g) corresponding to					
	Noncancer short-term[a] acceptable intake μg/day[b]		Cancer risk specific doses (μg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	110-184[f]	800-1300	0.01-0.02	0.1-1.0	1.0-2.0	22-360
- Soil ingestion[d], inhalation[e]	550-800	4000-5000	0.02-0.07	0.2-0.7	1.0-7.0	120-550
- Inhalation only[e]	vs[g]	vs	0.02-0.08	0.2-0.8	1.0-8	620-770
0.1 km from contaminated site - Inhalation only[e]	vs	vs	1-8	620-770	vs	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	vs	vs	vs	vs

[a] Short-term ≅ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] Ranges result in each case because 1) four PCBs (1242, 1248, 1254, 1260) are considered, each with a different vapor pressure, and 2) high and low values for soil-air partition coefficient are used in the calculations.

[g] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquids for the limit.

The symbol "vs" indicates that no upper-bound limit for PCB concentrations in soil can be derived from the exposure evaluation. This results mainly for two reasons. First, the emission rate cannot exceed the upper-bound value which can be expected when the air phase concentration of PCBs at the contaminated soil surface is maintained at the saturation point. The concentration at the saturation point corresponds to the vapor pressure concentration. Second, when the cover is applied, the emission rate is not only retarded, but also the concentration of PCBs in soil being ingested is controlled by the amount of PCBs adsorbed on soil in equilibrium with the air phase being emitted. Hence, the concentration of PCBs in the initially clean soil material cannot exceed the saturation point concentration. The PCB concentrations in soil corresponding to vapor saturation concentrations are 250, 55, 4, and 28 μg/g when $K_d = 1000$; and 10, 2.2, 0.2, and 1.1 when $K_d = 40$; for PCB-1242, PCB-1248, PCB-1254, and PCB-1260, respectively.

In actuality, the "no upper limit," or the level above vapor saturation, designated by vs, should be interpreted with great care. The assumptions used in the exposure evaluation are critical. They include, but are not limited to: 1) no soaking of clean cover by liquid PCBs for the thickness of 25 cm; 2) no disturbance of cover material by construction activities or children digging the ground; 3) no exposure to initial spills when the values applicable for 25-cm clean cover (Tables 23 through 26) are considered; 4) no population enters the area within the respective radius of distances from the site; and 5) the cover material is at least equivalent to soil material. The existence of free flowing PCBs liquids when placing clean soil cover material will have an effect of wetting the cover, resulting in the reduction of effective cover thickness. Hence, Assumption 1 will be tantamount to requiring the presence of no free liquids in the contaminated soil.

Since the ranges shown in these tables are dependent upon the values of the soil-air coefficient, the site-specific or contaminant-specific information will help find an appropriate level of PCBs for that particular condition. This can be done either by using the procedure outlined in the main body of the report, or can be conveniently done by looking up the values listed in the Appendix for each Aroclor at low and high values of soil-air partition coefficient.

The results in Tables 23 through 26 for each Aroclor assume that the 25-cm clean cover material is placed on top of contaminated soil. In this case, the intake rate by exposure to soil ingestion is calculated based on the estimated concentration profile existing in the cover material. This profile exists because of the establishment of the vapor-solid adsorption equilibrium between the vapors being emitted and the soil. The concentration profile, which changes as a function of time, is estimated by mathematical models. the concentration used for soil ingestion is the average concentration along the thickness of the initially clean cover material.

If the prevailing contaminants at a site are PCB-1242, for example, Table 19 can be interpreted as follows:

(1) When the site is amenable to access by children with possibilities of ingesting the contaminated soil exposed to the atmosphere, the permissible PCB concentrations levels in soil should range from 55 to 60 μg/g, and 92 to 247 μg/g for prevention of noncancer effects from 10-day exposures at soil ingestion rates of 3 g/day and 0.6 g/day, respectively.

When the site is accessible to children and the population has the potential of on-site exposures to the contaminated soil and air over a lifetime, the permissible PCB levels in soil should range from 0.008 to 0.01, 0.08 to 0.1, 0.8 to 1.0, and 8 to 13 μg/g, corresponding to the best estimate of an upper-bound oncogenic risk at 10^{-7}, 10^{-6}, 10^{-5} and 10^{-4}, respectively. The specific level will be dependent upon the likely soil ingestion rate and the extent of soil-air partitioning. Because of the PCB concentration profile being established in the soil column as volatilization occurs, the PCB concentration averaged over the depth will gradually decrease over time. Hence, if the population is allowed to enter the site at some time after site cleanup, the permissible levels for preventing 10-day noncancer health effects can change. Again, the specific level will be dictated by site-specific characteristics such as the soil-air partition coefficient.

(2) If there is no possibility of population entering the contaminated site within a radius of 0.1 km from the site, the PCB levels in the soil can remain at the no theoretical upper-bound limit value (vs μg/g) without exceeding the 10-day AI upon inhalation exposure for 10 days; and at 110-200 μg/g without exceeding the average daily dose corresponding to a 10^{-6} risk for lifetime exposure. Similar interpretations can be made for the results applicable to the carcinogenic risk listed at 10^{-4}, 10^{-5}, and 10^{-7}, and to sites without affected population up to 1 km from the site.

20. LIMITATIONS OF APPLICATION

It is assumed that the 25-cm (10-inch) clean cover material used remains undisturbed in the process of human activities on the site. At times this assumption may be found arbitrary, because an opportunity could exist that would expose the contaminated soil surface in contact with the atmosphere by inadvertent disturbances of soil surfaces, construction activities, utility installation, precipitation, or children playing on the site, to name a few. In this case, additional thickness of cover material should be used, or the site should be made inaccessible to children or should be kept from any activities that would lead to disturbance of the soil surfaces. Spills on top of the clean cover will result in a situation equivalent to the surface contamination case, requiring a more stringent concentration limit in soil. In this case, the results given for the 25-cm-thick clean cover material do not apply.

The tabulated results are intended to be applicable under certain specific conditions. Under conditions similar to those used in preparing the tables, the values can be used without additional evaluations. A particular situation may warrant a site-specific evaluation which may require the use of conditions different from what has been assumed in preparing the tables. If the analysis is available to show the specific type of Aroclor contaminating the soil, the individual table should be used. If the value for the soil-air partition coefficient can be better defined, the range of the permissible PCB concentration should be further narrowed.

21. REFERENCES

Binder, S. (1985) Estimating the amount of soil ingested by young children through trace elements. Report by the Centers for Disease Control.

Bruce, D.B. (1969) Workbook of atmospheric dispersion estimates, U.S. Department of Health, Education, and Welfare. Pub. No.: 999-AP-26.

Burkhard, L.P.; Armstrong, D.E.; Andren, A.W. (1985) Henry's Law constants for the polychlorinated biphenyls. Environ. Sci. Technol. 7:590-596.

Buser, H.R.; Rappe, C. (1979) Formation of polychlorinated dibenzofurans from the pyrolysis of individual PCB isomers. Chemosphere 8:157.

Carey, A. (Undated) Chemical/element concentrations in surface soils of selected U.S. urban areas. U.S. Environmental Protection Agency, private communication.

Carter, J.W. (1983) Onset of hepatomegaly in PCB (Aroclor 1254)-treated rats. Bull. Environ. Contam. Toxicol. 31(2):183-187.

Collins, W.T.; Capen, C.C. (1980a) Biliary excretion of thyroxine-I-125 and fine structural alterations in the thyroid glands of gunn-rats fed PCBs. Lab. Invest. 43:158.

Collins, W.T.; Capen, C.C. (1980b) Ultrastructural and functional alterations of the rat thyroid gland produced by polychlorinated biphenyls compared with iodide excess and deficiency, and thyrotropin and thyroxine administration. Virchows Arch. B. 33(3):213-231.

Collins, W.T.; Capen, C.C. (1980c) Fine structural lesions and hormonal alterations in thyroid glands of perinatal rats exposed in utero and fed by the milk to polychlorinated biphenyls. Am. J. Pathol. 99(1):125-141.

DuPont, R.R. (1985, Nov.) Evaluation of air emission release rate model predictions of hazardous organics from land treatment facilities. Presented at American Institute of Chemical Engineers meeting, Chicago, IL.

Farmer, W.J.; Yang, M.-S.; Letey, J., Dept. of Soil and Environmental Sciences, University of California-Riverside; Spenser, W.F., Science and Education Administration, Federal Research, USDA. (1980) Land disposal of hexachlorobenzene wastes: Controlling vapor movement in soil. EPA-600/2-80-119. Prepared for U.S. Environmental Protection Agency, Municipal Environmental Research Laboratory, Cincinnati, OH.

Grant, D.L.; Phillips, W.E.J. (1974) The effect of age and sex on the toxicity of Aroclor 1254, a polychlorinated biphenyl, in the rat. Bull. Environ. Contam. Toxicol. 12:145-152.

Hwang, S.T. (1982) Toxic emissions from land disposal facilities. Environ. Prog. 1:46.

Hwang, S.T. (1985, May) Assessing exposure to ground water contaminants migrated from hazardous waste facilities. Proceedings, Conference on Management of Unconfined Hazardous Waste Sites, Cincinnati, OH.

Hutzinger, O.; Safe, S.; Zitko, V., eds. (1974) The chemistry of PCBs. CRC Cleveland, OH: CRC Press.

Jury, W.A.; Spencer, W.F.; Farmer, W.J. (1983) Behavior assessment model for trace organics in soil. I. Model description. J. Environ. Qual. 4:558-564.

Karickhoff, S.W. (1979) Sorption of hydrophonic pollutants on natural sediments. Water Res. 13:241-248.

Kenaga, E.E.; Goring, C.A.I. (1980) Relationship between water solubility, soil sorption, octanol-water partitioning, and bioconcentration of chemicals in biota. In: Aquatic Toxicology. (Eaton, J.C.; Parnish, P.R.; Hendricks, A.C.; eds.). American Society for Testing and Materials, in press. ASTM STP 707.

Kimbrough, R.D.; Falk, H.; Stehr, P. (1984) Health implications of 2,3,7,8-tetrachloro-dibenzodioxin (TCDD) contamination of residual soil. J. Toxicol. Environ. Health 14:47.

Kimbrough, R.D.; Squire, R.A.; Linder, R.E.; Strandberg, J.D.; Montali, R.J.; Burse, V.W. (1975) Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. J. Natl. Cancer Inst. 55:1453-1459.

Lepow, M.L. (1975) Investigations in sources of lead in the environment of urban children. Environ. Res. 10:415.

Lorenz, H.; Neumeier, G. (1983) Polychlorinated biphenyls: profile of a group of substances. MMV Medizin Verlag München.

MacKay, D.; Leinonen, P.L. (1975) Rate of evaporation of low-solubility contaminants from water bodies to atmosphere. Env. Sci. Technol. 9:1178.

Martin, D.O. (1976) The change of concentration standard deviation with istance. J. Air Poll. Control Assoc. 26:145.

Monsanto Chemical Company. (Undated) The AROCLORS--physical properties and suggested applications.

MRI. (1984, December) Thermal degradation products from dielectric fluid. Prepared for U.S. Environmental Protection Agency, Office of Toxic Substances. EPA-560/5-84-009.

National Institute for Occupational Safety and Health (NIOSH). (1977) Criteria for a recommended standard: occupational exposure to polychlorinated biphenyls (PCBs). DHEW (NIOSH) Pub. No. 77-225. U.S. Government Printing Office, Washington, D.C.

New York State Department of Environmental Conservation. (1979) New York State air quality report, continuous and manual air monitoring system. DAR-80-1. Albany, NY.

New York State Department of Health. (1981, March 16) Memorandum from John Hawley.

Nisbet, I.C.T.; Sarofim, A.F. (1972) Rates and routes of transport of PCBs in the environment. Environ. Health Perspect. 1:21.

Roels, H.A.; Buchet, J.-P.; Lauwerys, R.R.; Bruaux, P; Clyaeys-Thoreau, F.; Lafontaine, A.; Verduyn, G. (1980) Exposure to lead by the oral and the pulmonary routes of children living in the vicinity of a primary lead smelter. Environ. Res. 22:81-94.

SCS Engineers. (Undated) W-E-T model hazardous waste data base. Final draft. Prepared for U.S. Environmental Protection Agency, Office of Solid Waste, Washington, D.C. Reston, VA.

Snyder, W.S. (1975) Report of the task group on reference manual. International Commission of Radiological Protection No. 23. Pergamon Press, New York, NY.

Thibodeaux, L.J.; Hwang, S.T. (1982) Landfarming of petroleum wastes: modeling the air emission problem. Environ. Prog. 1:42-46.

University of Wisconsin-Madison, Water Resources Center and Water Chemistry Program. (1980, March) Atmospheric chemistry of PCBs and PAHs. Prepared for U.S. Environmental Protection Agency, Chicago, IL.

U.S. Environmental Protection Agency. (Undated) Guideline for assessing human exposure. Office of Solid Waste.

U.S. Environmental Protection Agency. (1976a, January) Development of a study plan for definition of PCB usage, wastes, and potential substitution in the investment casting industry. NTIS No.: PB251842 C.1.

U.S. Environmental Protection Agency, Office of Toxic Substances. (1976b, February) PCBs in the United States: industrial use and environmental distribution; Task 1. Prepared by Versar, Inc. NTIS No.: PB252402.

U.S. Environmental Protection Agency. (1976c, March) National Conference on Polychlorinated Biphenyls. NTIS No.: PB253248.

U.S. Environmental Protection Agency. (1978, March) Microeconomic impacts of the proposed PCB ban regulations. NTIS No.: PB281881 C.1.

U.S. Environmental Protection Agency. (1979a) Water-related environmental fate of 129 priority pollutants. Vol. 1. EPA-440/4-79-029a.

U.S. Environmental Protection Agency. (1979b, May) Polychlorinated biphenyls 1929-1979: Final report. Office of Toxic Substances. NTIS No.: PB256559.

U.S. Environmental Protection Agency. (1980a) Attenuation of water-soluble polychlorinated biphenyls by earth materials. EPA-600/2-80-027.

U.S. Environmental Protection Agency. (1980b) Ambient water quality criteria document for PCBs. Environmental Criteria and Assessment Office, Cincinnati, OH. EPA-440/5-80-068.

U.S. Environmental Protection Agency. (1980c, April) Determination of Henry's Law constants of selected priority pollutants. Municipal Environmental Research Laboratory, Cincinnati, OH. (Authors: Warner, H.P.; Cohen, J.M.; Ireland, J.C.)

U.S. Environmental Protection Agency. (1981a, April) Evaluation of PCB destruction efficiency in an industrial boiler. EPA-600/2-81-055a.

U.S. Environmental Protection Agency. (1981b, May) Environmental impact statement on the Hudson River PCB Reclamation Demonstration Project. Region II, New York, NY.

U.S. Environmental Protection Agency. (1983) Analysis of adipose and blood sera samples for individual PCB isomers. Draft Final Report on EPA Contract 68-01-5915, 11/30/81 - 5/31/83.

U.S. Environmental Protection Agency. (1984a, Nov.) Risk analysis of TCDD-contaminated soil. EPA-600/8-84-031.

U.S. Environmental Protection Agency. (1984b) Code of Federal Regulations, Title 40, Part 761.

U.S. Environmental Protection Agency. (1985a) Drinking water criteria document for polychlorinated biphenyls (PCBs). Environmental Criteria and Assessment Office, Cincinnati, OH. Final Draft. EPA-600/X-84-198-1.

U.S. Environmental Protection Agency. (1985b, June 12) Personal communication, Don R. Clay, Director, Office of Toxic Substances.

U.S. Environmental Protection Agency. (1985c, Nov. 7) Personal communication, Martin, P. Halper, Director, Exposure Evaluation Division, Office of Toxic Substances.

U.S. Environmental Protection Agency. (1985d, Aug.) Development of statistical distributions or ranges of standard factors used in exposure assessments. Prepared by GCA Corporation for the Office of Health and Environmental Assessment. NTIS No: PB85-242667.

U.S. Environmental Protection Agency. (1985e) Personal communication with Office of Toxic Substances.

U.S. Food and Drug Administration. (1984) Code of Federal Regulations, Title 21, Part 109.30.

Veith, G.D.; Macek, K.J.; Petrocelli, S.R.; Carroll, J. (1980) An evaluation of using partition coefficients and water solubility to estimate bioconcentration factors for organic chemicals in fish. J. Fish. Res. Board Can. (Prepublication copy).

Versar, Inc. (1976) Assessment of wastewater management, treatment technology, and associated costs for abatement of PCB concentrations in industrial effluents. Prepared for U.S. Environmental Protection Agency, Office of Toxic Substances. NTIS No.: PB251433 C.1.

Versar, Inc. (1977) A first-order mass balance model for the sources, distribution, and fate of PCBs in the environment. Prepared for the U.S. Environmental Protection Agency, Office of Toxic Substances, Washington, D.C.

Villeneuve, D.C.; Grant, B.L.; Khera, K.; Clegg, D.J.; Baer, H.; Phillips, W.E.J. (1971) The fetotoxicity of a polychlorinated biphenyl mixture (Aroclor 1254) in the rabbit and in the rat. Environ. Physiol. 1:67-71.

Wark, K.; Warner, C.F. (1981) Air pollution: its origin and control. New York: Harper and Row.

Wedeen, R.P.; Mailik, D.K.; Baiuman, V.; Bogden, J.D. (1978) Geophagic lead nephropathy: case report. Environ. Res. 17:409.

APPENDIX A

MODELS USED IN AIR RELEASE RATE CALCULATIONS

DERIVATION OF MODELS FOR ESTIMATING VOLATILE EMISSIONS FROM CONTAMINATED SOIL COLUMNS UNDER TRANSIENT CONDITIONS

Because of the limited aqueous solubility and high soil affinity of PCBs, it has been assumed that these compounds move vertically in soils, primarily by diffusion in the vapor phase. If transport of PCBs is by vapor phase diffusion through interstitial spaces between soil particles, a mass balance over an infinitesimal vertical element of soil can be written as follows:

$$AE\left(-D_{ei}\frac{\partial C}{\partial z}\right)_{z,t} - AE\left(-D_{ei}\frac{\partial C}{\partial z}\right)_{z+\Delta z,t} = A\Delta z\frac{\partial C}{\partial t} + A\Delta z\overline{P}_s\frac{\partial C_s}{\partial t} \qquad \text{(A-1)}$$

where:

A = cross-sectional area of interest, cm^2
C = concentration of PCBs in the vapor phase in soil pores, g/cm^3
C_s = concentration of PCBs in soil, g/g
D_i = molecular diffusivity, cm^2/s
D_{ei} = effective diffusivity, cm^2/s ($=Di \cdot E^{1/3}$)
E = pore porosity
$\overline{P}_s$ = bulk density of soil = true density of soil, Ps, multiplied by (1-E), g/cm^3
t = time, seconds
z = depth measured from the soil-air interface, cm

In Eq. (A-1), the effective diffusivity, D_{ei}, is used in place of $E^{1/3} \cdot D_i$ to account for the tortuosity effect in porous media. The use of effective diffusivity is consistent with the findings which describe emission rates of volatile chemicals from landfills and soils (Hwang, 1982; Thibodeaux, 1979; Farmer et al., 1980). The effective porosity for dry soil is used for simplicity. The effect of moisture can be incorporated in the porosity term as shown by Farmer et al. (1980).

Since changes in soil and vapor phase PCB concentrations occur slowly, it can be assumed that vapor phase concentrations and soil concentrations of PCBs are in local equilibrium. If PCB concentrations in soil and in interstitial vapors approach equilibrium, they are related by the following equation:

$$C_s = \frac{K_d}{H} \cdot C \qquad \text{(A-2)}$$

where

K_d = soil/water partition coefficient

H = Henry's constant

Rearranging Eq. (A-1) and substituting Eq. (A-2) into the resulting relationship yields

$$\mathrm{Dei}\ \frac{\partial^2 C}{\partial z^2} = \left(1 + \frac{\bar{P}_s \cdot K_d}{E \cdot H}\right) \frac{\partial C}{\partial t} \qquad \text{(A-3)}$$

or

$$\alpha \frac{\partial^2 C}{\partial z^2} = \frac{\partial C}{\partial t} \qquad \text{(A-3)}$$

where

$$\alpha = \frac{D_{ei} . E}{(E + P_s \cdot [1 - E] \cdot K_d / H)} \qquad \text{(A-4)}$$

α can also be defined as

$$\alpha = \frac{D_{ei}}{1 + K \cdot S} \qquad \text{(A-5)}$$

where

$$K = \frac{K_d}{H} \cdot \rho_s$$

Eq. (A-3) can be solved to estimate PCB soil concentration, vapor phase concentration, and emission rate into air above soil for the various cases described in this report if initial and boundary conditions are specified for each of these cases.

Case 1. Surface is exposed to the atmosphere. The boundary and initial conditions are

1. I.C. $C = (H/K_d)C_{SO}$, at $t = 0$, $z > 0$
2. B.C. $C = (H/K_d)C_{SO}$, at $z = \infty$, $t > 0$
3. B.C. $C = 0$, at $z = 0$, $t > 0$

where C_{SO} is the initial concentration of PCBs in soil. The solution to Eq. (A-3) for the above initial and boundary conditions is

$$C = (H/K_d)C_{SO} \cdot \mathrm{erf}\left(\frac{z}{2\sqrt{\alpha t}}\right) \tag{A-6}$$

where

$$\mathrm{erf}\ (n) = \text{error function} = \frac{2}{\sqrt{\pi}} \int_0^n \exp(-n^2)\ dn$$

The flux rate at the soil-air interface (N_A) can be estimated as a function of time from equation (A-6) by using the concentration gradient as follows:

$$N_A = -E \cdot D_{ei} \left.\frac{\partial C}{\partial z}\right|_{z=0} = \frac{E\ D_{ei}}{\sqrt{\Pi \alpha t}} \cdot \frac{H}{K_d} \cdot C_{SO} \tag{A-7}$$

The boundary conditions used here are superior to those used by Jury et al. (1983), assuming that the vapor-phase boundary layer is rate-controlling. Experiments by DuPont (1985) on emission rates from contaminated soil show that when the emission rates for volatile organics are plotted against the reciprocal of $\sqrt{t}$, a straight line is obtained. This observation is consistent with the relationships given by Eq. (A-7), and Thibodeaux and Hwang (1982). The model derived by Jury et al. (1983), based on the boundary conditions of the controlling boundary layer in the air phase, does not provide a straight-line relationship between emission rate and $1/\sqrt{t}$. For this reason, the relationship derived in this report is used for exposure evaluation.

The average flux rate, N_A, over an exposure interval, T, can be calculated using Eq. (A-7).

$$\overline{N_A} = \frac{\int_0^T N_A\, dt}{T} = \frac{2 \cdot E \cdot D_{ei}}{\sqrt{\Pi \alpha T}}\ \frac{H}{K_d}\ C_{SO} \tag{A-8}$$

or

$$\overline{N}_A(T) = 2\ N_A(T) \tag{A-9}$$

To estimate the total average emission rate, Q, the flux rate defined in Eq. (A-9) must be multiplied by the area of soil contaminated.

$$Q = A \cdot \overline{N}_A \qquad \text{(A-10)}$$

Furthermore, while Eq. (17) in Section 16 can be used at any distance X from the site to estimate air concentrations of PCBs, it cannot be used on-site. Although at present there is no generally accepted methodology for estimating on-site concentrations from an area source, on-site PCB air concentration was estimated based on a "box model" approach, by using the equation

$$C = \frac{Q}{LS \cdot V \cdot H} \qquad \text{(A-11)}$$

where

H = mixing height = 2 m

V = average wind speed within mixing zone

= 0.5 wind speed at the mixing height = 0.5 x 4.5 meter/sec = 2.25 m/s

LS = width dimension of contaminated area perpendicular to the wind direction = $\sqrt{A}$ = 45 m

A need exists for development of a more rigorous approach to estimating on-site ambient air concentrations. Time constraints did not allow development and validation of a rigorous model.

Estimation of ingestion of contaminated soil required the calculation of an appropriate soil concentration. This concentration was calculated by determining the average concentration of PCB in soil to a depth of or 25.4 cm (10 inches) for a period of 6 years beginning at time 0. Because the error function has no closed-form solution it was approximated by

$$C_s = \frac{4\,C_{SO}}{\Pi} \sum_{n=0}^{\infty} \frac{e^{-\frac{\alpha(2n+1)^2}{L^2}\frac{\Pi^2 t}{4}}}{2n+1} \sin\left\{\frac{(2n+1)\Pi}{2}\frac{z}{L}\right\} \qquad \text{(A-12)}$$

where L is a depth which was selected such that

$$C_s\,(L,t) \approx C_{SO}$$

for all exposure durations. In calculations reported in this report, L was set equal to 250 cm. Integrating C_s of the exposure duration t_D (5 years) and depth ℓ (25 cm) yields an equation for average PCB soil concentration, $\overline{C_s}$

$$\overline{C_s} = \frac{1}{t_D \cdot \ell} \int_0^{t_D} \int_0^{\ell} C_s\,dz = \frac{32L^3\,C_{SO}}{\Pi^4 \alpha \ell t_D} \sum_{n=0}^{\infty} \left\{1 - \cos\left(\frac{2n+1}{2}\,\Pi\,\frac{\ell}{L}\right)\right\} \cdot \left\{\frac{1 - e^{-\alpha\frac{(2n+1)^2}{4L^2}\Pi^2 t_D}}{(2n+1)^4}\right\} \qquad \text{(A-13)}$$

Case 2. The contaminated surface is covered with PCB-free soil material. Let ℓ = thickness of cover, cm, and L = the depth of contamination measured from the top of cover material, cm. The initial and boundary conditions become:

1. I.C. $C = 0$, $0 < z < \ell$, at $t = 0$
2. I.C. $C = C_0$, $\ell < z < L$, at $t = 0$
3. B.C. $C = 0$, $z = 0$, at $t > 0$
4. B.C. $\frac{\partial C}{\partial z} = 0$, $z = L$, at $t > 0$

where C_0 is the initial concentration of PCBs in the vapor phase, which can be obtained by $C_0 = (H/K_d)C_{SO}$. Eq. (A-3) with these initial and boundary conditions can be solved using the Fourier Series technique. The solution is

$$C = \frac{4C_0}{\Pi} \sum_{n=0}^{\infty} e^{\frac{-\alpha(2n+1)^2 \Pi^2 t}{4L^2}} \cdot \sin\left\{\frac{2n+1}{2} \Pi \frac{z}{L}\right\} \cos\left\{\frac{2n+1}{2} \Pi \frac{\ell}{L}\right\} \tag{A-14}$$

The flux rate at the soil-air interface (N_A) can be estimated as a function of time from equation (A-14)

$$N_A = E \cdot D_{ei} \left.\frac{\partial C}{\partial z}\right|_{z=0} = \frac{2\ C_0\ E \cdot D_{ei}}{L} \sum_{n=0}^{\infty} e^{-\frac{\alpha\ (2n+1)^2\ \Pi^2\ t}{4L^2}} \cos\left\{\frac{(2n+1)}{2} \frac{\Pi\ell}{L}\right\} \tag{A-15}$$

The average emission rate over a time period, T, can be obtained by integration of Eq. (A-15). The result is

$$\overline{N_A} = \frac{8(H/K_d) \cdot C_{S0} \cdot E \cdot D_{ei} \cdot L}{\alpha \pi^2 T} \sum_{n=0}^{\infty} \frac{1}{(2n+1)^2} \left[e^{-\frac{\alpha\ (2n+1)^2\ \Pi^2)t_1}{4 \cdot L^2}} - e^{-\frac{\alpha\ (2n+1)^2\ \Pi^2)t_2}{4 \cdot L^2}} \right] \cdot \cos\left\{\frac{(2n+1)}{2} \frac{\Pi\ell}{L}\right\} \tag{A-16}$$

or

$$\overline{N_A} = \frac{8(H/K_d) \cdot C_{S0} \cdot E \cdot (1+K \cdot S) \cdot D_{ei}\ L}{\pi^2 T} \sum_{n=0}^{\infty} \frac{1}{(2n+1)^2} \left[e^{-\frac{D_i E^{1/3} (2n+1)^2\ \Pi^2)t_1}{(1+K \cdot S)L^2\ \cdot 4}} - e^{-\frac{D_i E^{1/3} (2n+1)^2\ \Pi^2)t_2}{(1+K \cdot S) \cdot L^2\ \cdot 4}} \right] \cos\left\{\frac{(2n+1)}{2} \frac{\Pi\ell}{L}\right\} \tag{A-17}$$

The summation of terms given in Eq. (A-16) can be conveniently carried out by means of computer simulation. The time interval t_2-t_1 should be set equal to the exposure interval. In calculating exposures, the maximum average exposure was estimated. This was achieved by calculating N_A as a function of time and determining the time at which the maximum value of N_A occurred; t_1 was then set equal to this time.

It should be noted that when the value of the expression $\frac{\alpha(2n+1)^2\Pi^2}{4L^2}$ in the exponential term of Eq. (A-16) is small or considerably less than 1, the average of the exponential term over a time, t, will be close to 1. In this situation, averaging of the exponential term of Eq. (A-15) by the integration formulae given by Eq. (A-16) or Eq. (A-17) may easily result in an erroneous answer because one has to evaluate very precise numbers of many decimal points for the values of the exponential term. It is more practical to numerically average Eq. (A-15) than to obtain the average value by using the integration formula given by Eq. (A-16) or Eq. (A-17):

$$\overline{N_A} = \frac{2(H/K_d)C_{SO}\cdot E\cdot D_{ei}}{L}\,\frac{1}{T}\sum_{n=0}^{\infty}\left[\int_{t_1}^{t_2} e^{-\frac{\alpha\,(2n+1)^2\,\Pi^2 t}{4\cdot L^2}}\right] dt\cdot\cos\left\{\frac{(2n+1)}{2}\frac{\Pi \ell}{L}\right\} \tag{A-18}$$

The steps of the summation and the integration with respect to n and t, respectively, need to be carried out by means of a computer.

As in Case 1, Eqs. (A-10) and (A-11) are used to estimate emissions rate and on-site air concentration of PCBs. However, Eq. (A-17) or (A-18) is substituted into Eq. (A-10) as an estimate of flux rate.

Also as in Case 1, the average soil concentration used to estimate ingestion of soil must be calculated. This can be accomplished by noting that C_{SO}

$= C_0 \cdot K_d/H$, substituting this relationship into Eq. (A-14) and integrating the resulting equation over the depth interval ℓ and over the time interval t_1 to $t_1 + t_D$. The result is

$$\overline{C_s} = \frac{1}{t_D \ell} \int_{t_1}^{t_1+t_D} \int_0^{\ell} C_s \, dz = \frac{32 L^3 C_{SO}}{\Pi^4 \alpha \ell t_D} \sum_{n=0}^{\infty} \left\{1 - \cos \frac{(2n+1)}{2} \frac{\Pi \ell}{L}\right\} \left\{ e^{-\frac{\alpha (2n+1)^2 \Pi^2)}{4 \cdot L^2} t_1} - e^{-\frac{\alpha (2n+1)^2 \Pi^2}{4 \cdot L^2} (t_1 + t_D)} \right\} \frac{\cos \left\{\frac{(2n+1)}{2} \frac{\Pi \ell}{L}\right\}}{(2n+1)^4} \qquad \text{(A-19)}$$

where t_D = 5 years and ℓ = 25 cm.

When the initial PCB soil concentration used in estimating exposures exceeds the concentration at which the vapor pressure of PCB is achieved, a different model must be used in both Case 1 and Case 2. The vapor phase PCB concentration that can be achieved in the interstitial voids in soil is limited to the concentration corresponding to the vapor pressure. While this limits the emission rate, it should be noted that as the soil zones nearest the air-soil interface become depleted of PCB, the emission rate decreases. If PCB is present in soil concentrations that produce the vapor pressure in the vapor phase, the average emission rate may be increased because soil near the surface is depleted less rapidly.

In modeling this phenomenon it has been assumed that at any given time, the concentration profile of PCB in soil as a function of depth is a steady-state profile. As in the previous models, the concentration of PCB in the interstitial soil void space is assumed to be in equilibrium with PCB soil concentrations. Given these assumptions and the initial conditions that

$$C_0 = \frac{H \cdot C_{ss}}{K_d}$$

$$C_s = C_{SO}$$

where

C_{ss} = PCB soil concentration at which the vapor pressure is achieved.

A mass balance can be written to determine the rate of depletion from soil. If the soil concentration profile is as defined in Figure A-1, this mass balance is

$$\frac{dz}{dt} = \frac{E \cdot D_{ei} \cdot \frac{\partial C}{\partial z}}{\frac{1}{2} \{ C_{ss}[P(1-E)+E \cdot K_d/H]\} + P_s(1-E)(C_{SO}-C_{ss})} \qquad \text{(A-20)}$$

Because we assume that any any time the soil and vapor PCB approach their steady-state concentrations,

$$\frac{\partial C}{\partial z} = \frac{C_0}{z} \qquad \text{(A-21)}$$

substituting equation (A-21) into equation (A-20) and integrating the resulting equation over the time interval 0 to t and the corresponding depth interval 0 to z yields the result

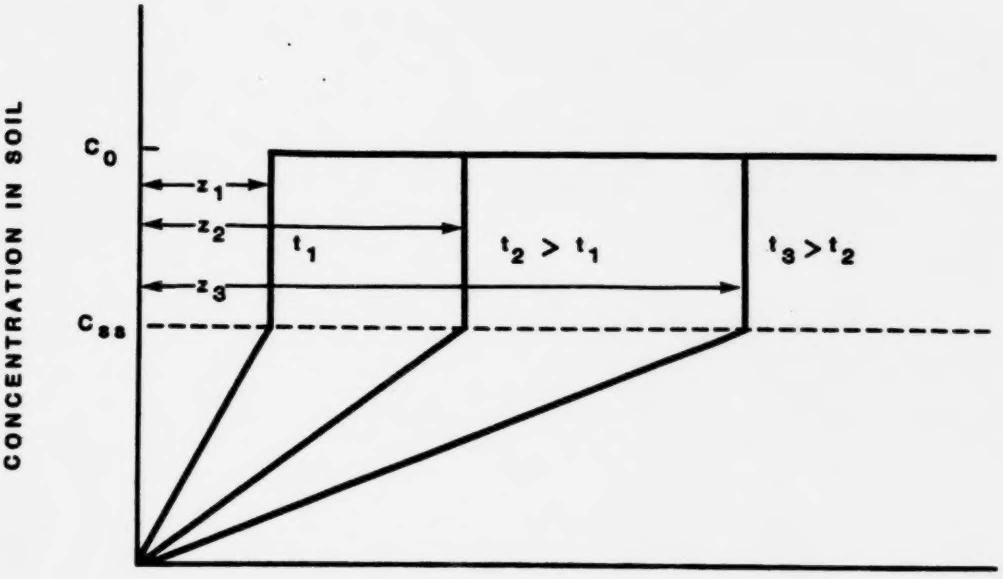

CONCENTRATION IN SOIL
C_0
C_{ss}
z_1
z_2
z_3
t_1
$t_2 > t_1$
$t_3 > t_2$
DEPTH (z)

Figure A-1. Model of chemical vapor movement through soil when partial pressure is equal to vapor pressure.

$$z = 2\sqrt{\frac{E\cdot D_{ei}\cdot C_{ss}\cdot t}{E\cdot C_{ss} + 2\cdot P\cdot(1-E)\cdot C_{s0}\cdot K_d/H - (1-E)\cdot P\cdot C_{ss}\cdot K_d/H}} \qquad \text{(A-22)}$$

As in the previous case, the flux rate can be calculated as

$$N_A = E\cdot D_{ei}\frac{\partial C}{\partial x} = E\cdot D_{ei}\frac{C_0}{z} = \frac{E\cdot D_{ei}\cdot C_{ss}\cdot H}{z\quad K_d} \qquad \text{(A-23)}$$

or

$$N_A = \frac{\sqrt{E\cdot D_{ei}\cdot C_{ss}} + \{2\cdot(1-E)\cdot P\cdot C_{s0} + E\cdot C_{ss}\cdot H/K_d - (1-E)\cdot P\cdot C_{ss}\}}{2\sqrt{t\cdot K_d/H}} \qquad \text{(A-24)}$$

If the average flux is determined for the time interval T, it is easy to show that

$$\overline{N}_A(T) = 2N_A(T) \qquad \text{(A-25)}$$

As indicated previously, Eqs. (A-10) and (A-11) can be used to estimate emission rate and on-site air concentrations. However, Eq. (A-24) is substituted into Eq. (A-10) as an estimate of flux rate in this case.

The average soil concentration to a depth of 25 cm over the exposure duration of up to 5 years of exposure must be determined to estimate ingested dose of PCBs. The equation used to estimate this average depends on whether the depth z in Eq. (A-22) is less or greater than L_1 (25 cm) at the end of the ingestion exposure periods. The time T_5 at which $z = L_1$ is easily calculated using the following equation:

$$T_5 = 0.25\cdot L_1{}^{2}\cdot(E\cdot C_{ss} - 2(1-E)P\cdot C_{SO}\cdot K_d/H - P\cdot(1-E)\cdot C_{ss}\cdot K_d/H)/(E\cdot D_{ei}\cdot C_{ss}) \qquad \text{(A-26)}$$

If the ingestion exposure period, T, is less than T_5, the depth, z, will always be less than L_1, and the average soil PCB concentration, C_s, can be calculated as follows:

$$\overline{C}_S = 2\cdot(C_{ss}-2\cdot C_{SO})\ \{E\cdot D_{ei}\cdot C_{ss}\cdot T/[E\cdot C_{ss}+2(1-E)\cdot P\cdot C_{SO}\cdot K_d/H - (1-E)\cdot P\cdot C_{ss}\cdot K_d/H]\}^{1/2}/3L_1 + C_{SO} \qquad \text{(A-27)}$$

If the ingestion exposure period is greater than T_5, the depth, z, will be greater than L_1 at the end of the exposure period, and the average soil PCB concentration can be calculated as follows:

$$\begin{aligned}\overline{C}_S = {} & 2\cdot(C_{ss}-2\cdot C_{SO})\ \{E\cdot D_{ei}\cdot C_{ss}/[E\cdot C_{ss}+2(1-E)\cdot P\cdot C_{SO}\cdot K_d/H \\ & - (1-E)\cdot C_{ss}\cdot P\cdot K_d/H]\}^{0.5}\cdot T_5{}^{1.5}\ /(3\cdot L_1\cdot T) + C_{SO}\cdot T_5/T \\ & + 2\cdot(C_{ss}-L_1)\ \{[E\cdot C_{ss}+2(1-E)\cdot P\cdot C_{SO} - (1-E)\cdot P\cdot C_{ss}\cdot K_d/H]\ / \\ & [E\cdot D_{ei}\cdot C_{ss}]\}^{.5}\ \{T^{0.5} - T_5{}^{0.5}\}/T\end{aligned} \qquad \text{(A-28)}$$

When clean cover is placed over contaminated soil, a similar model can be developed as in the case where soil is contaminated to the surface. For such situations, assuming that local equilibrium between vapor and solid phases and steady-state concentration distributions at any time are attained, the following mass balance which yields relationships illustrated in Figures A-2 and A-3, which define the concentrations of PCB in soils as a function of depth

$$L_4 = \{2 \cdot L_1 \cdot (1-E) \cdot P(C_{SO}-C_{ss}) + \{4 \cdot [(1-E) \cdot L_1 \cdot (C_{SO}-C_{ss}) \cdot P]^2$$

$$+ 4L_1^{2} \cdot (E \cdot H/K_d + (1-E) \cdot P) \cdot C_{ss} \cdot [2(1-E) \cdot P \cdot (C_{SO}-C_{ss}) + E \cdot H/K_d + (1-E) \cdot P) \cdot C_{ss})]\}^{0.5}$$

$$/ \{2 \cdot [2 \cdot (1-E) \cdot P \cdot (C_{SO}-C_{ss}) + [E \cdot H/K_d + (1-E) \cdot P] \cdot C_{ss}]\} \qquad \text{(A-29)}$$

The time at which PCB reaches the air-soil interface, T_b, can be estimated by rearranging Eq. (A-22) and substituting $L_4 + L_1$ for z, as follows:

$$T_b = (L_4+L_1)^{2} \cdot \{E \cdot C_{ss} + 2 \cdot (1-E) \cdot P \cdot C_{SO} \cdot K_d/H - (1-E) \cdot P \cdot C_{ss} \cdot K_d/H\}$$

$$\cdot \{4 \cdot E \cdot D_{ei} \cdot C_{ss}\}^{-1} \qquad \text{(A-30)}$$

Integrating Eq. (A-24) over the exposure time interval T_b to $T+T_b$ and dividing the result by T yields the following expression for the average flux over the exposure period:

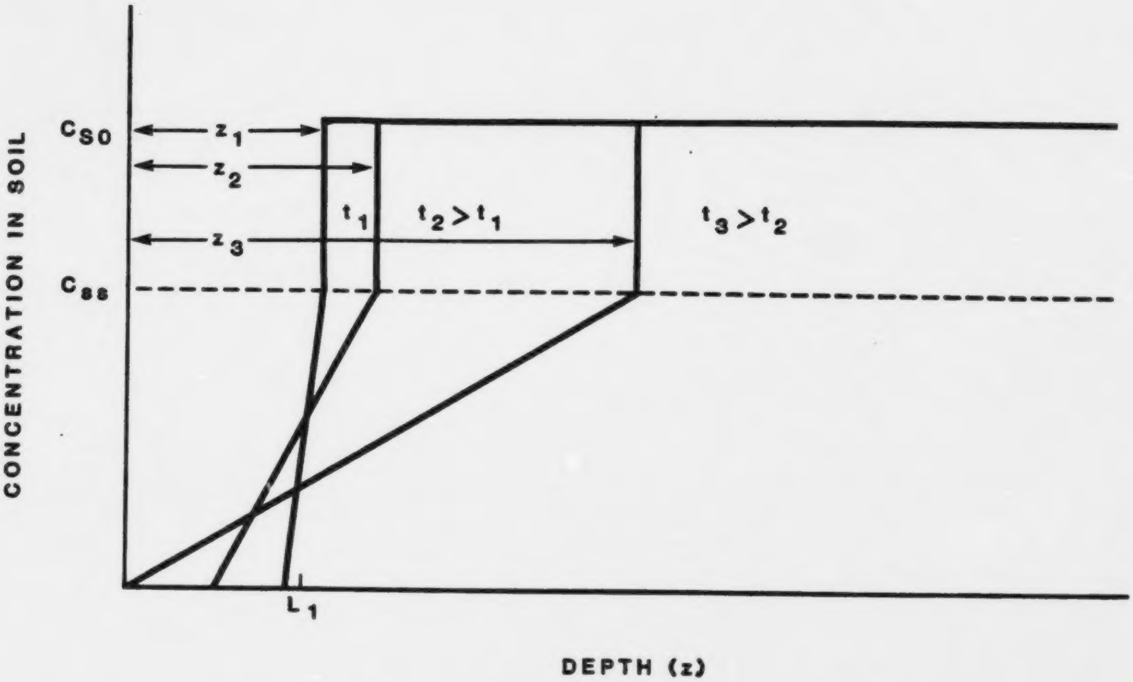

CONCENTRATION IN SOIL
C_{S0}
C_{ss}
z_1
z_2
z_3
t_1
$t_2 > t_1$
$t_3 > t_2$
L_1
DEPTH (z)

Figure A-2. Model of chemical vapor movement through soil when partial pressure is equal to vapor pressure.

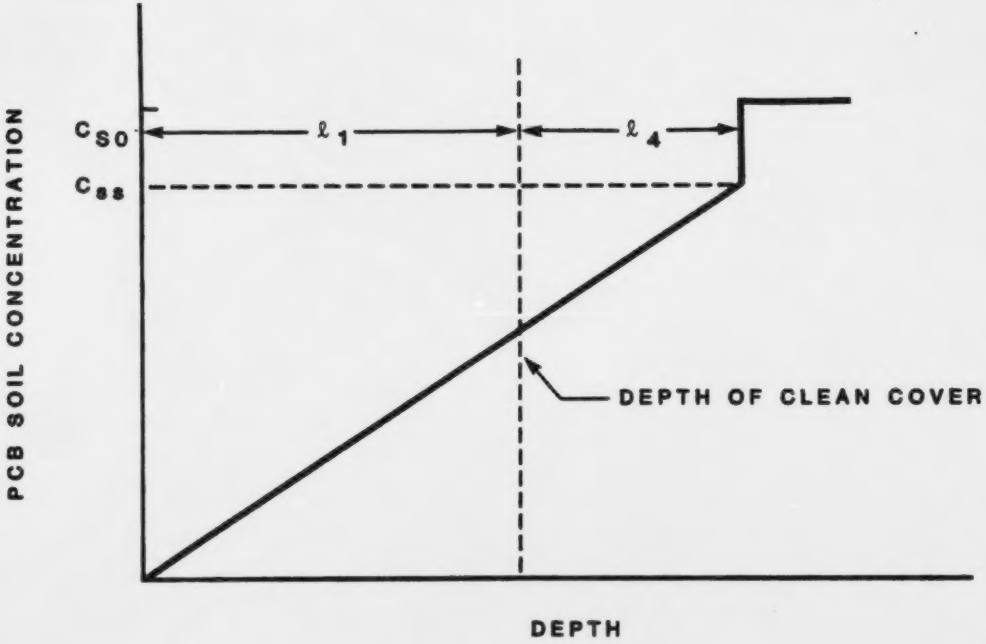

PCB SOIL CONCENTRATION
C_{SO}
C_{ss}
ℓ_1
ℓ_4
DEPTH OF CLEAN COVER
DEPTH

Figure A-3. Mass balance for vapor movement through soil when partial pressure is equal to vapor pressure.

$$\overline{N_A} = \frac{1}{T}\int_{T_b}^{T_b+T} N_A \, dt =$$

$$\left\{ \frac{\surd\, E\cdot D_{ei}\cdot C_{ss}\ \{2\cdot(1-E)\cdot P\cdot C_{SO} + E\cdot C_{ss}\cdot H/K_d - (1-E)\cdot P\cdot C_{ss}}{\surd\, K_d/H} \right\}$$

$$\cdot \left\{ \frac{(T_b+T)^{1/2} - T_b^{1/2}}{T} \right\} \qquad \text{(A-31)}$$

As before, Eqs. (A-10) and (A-11) can be used to estimate emission rates and on-site air concentrations.

Finally, the average soil concentration to a depth of 25 cm over the exposure of duration up to 5 years must be determined in order to estimate ingested dose of PCBs. The equation used to estimate this average is:

$$\overline{C}_s = C_{ss}\cdot L_1\cdot T_5^{1/2}\{(T_5+T)^{1/2} - T_5^{1/2}\} \ /\{T\cdot(L_1+L_4)\} \qquad \text{(A-32)}$$

Calculation of the Depth-Averaged Concentration for Uncovered Surface

We want to find the average concentration of PCBs in soil over the exposure period. As time progresses, the concentration in soil decreases because of volatilization. First, we want to find the time when the emission rate at any time equals the average emission rate. We equate Eqs. (A-7) and (A-8). Then

$$\frac{E\cdot D_{ei}}{\sqrt{\Pi\alpha t}} \cdot \frac{H}{K_d} \cdot C_{SO} = \frac{2E\cdot D_{ei}}{\sqrt{\Pi\alpha T}} \cdot \frac{H}{K_d} \cdot C_{SO} \qquad \text{(A-33)}$$

The emission rate equals the average emission rate at t = T/4, where T is the exposure time (1 day, 10 days, or 70 years), and t is any time. From Eq. (A-6), the vertically-averaged concentration over a depth of 2 cm is:

$$\overline{C} = \frac{H}{K_d} \cdot C_{SO} \frac{1}{z} \int_0^z \text{erf}\left(\frac{z}{2\sqrt{\alpha t}}\right) dz \qquad \text{(A-34)}$$

or

$$\overline{C}_s = C_{SO} \frac{1}{z} \int_0^z \text{erf}\left(\frac{z}{2\sqrt{\alpha t}}\right) dz \qquad \text{(A-35)}$$

The integral should be numerically evaluated at t = T/4, T being the exposure period for developing health advisories, and at an appropriate depth within which the concentration average is desired.

REFERENCES (APPENDIX A)

DuPont, R.R. (1985, Nov.) Evaluation of air emission release rate model predictions of hazardous organics from land treatment facilities. Presented at American Institute of Chemical Engineers meeting, Chicago, IL.

Farmer, W.J.; Yang, M.-S.; Letey, J., Dept. of Soil and Environmental Sciences, University of California-Riverside; Spenser, W.F., Science and Education Administration, Federal Research, USDA. (1980) Land disposal of hexachlorobenzene wastes: controlling vapor movement in soil. EPA-600/2-80-119. Prepared for U.S. Environmental Protection Agency, Municipal Environmental Research Laboratory, Cincinnati, OH.

Hwang, S.T. (1982) Toxic emissions from land disposal facilities. Environ. Prog. 1:46.

Jury, W.A.; Spencer, W.F.; Farmer, W.J. (1983) Behavior assessment model for trace organics in soil. I. Model description. J. Environ. Qual. 4:558-564.

Thibodeaux, L.J. (1979) Chemodynamics. New York, NY: John Wiley and Sons.

Thibodeaux, L.J.; Hwang, S.T. (1982) Landfarming of petroleum wastes: modeling the air emission problem. Environ. Prog. 1:42-46.

APPENDIX B

EXAMPLE EMISSION RATE CALCULATIONS FOR FOUR STUDIED SCENARIOS

Example calculations for estimating volatile emissions of PCBs based on the models presented in Appendix A for unsteady-state conditions, and under steady-state conditions are presented below.

Case 1. Unsteady-state emission: no cover.

The contaminated soil is exposed to the atmosphere, and no clean soil cover is applied on top of the contaminated surface. PCB-1254 is used as an example. The average emission rate, which is obtained by averaging the instantaneous emission rate over a time period, t (sec), can be obtained by:

$$\overline{N}_A = \frac{H}{K_d}\left(\frac{g\ soil}{cm^3\ air}\right) E \frac{2\ D_{ei}\left(\frac{cm^2}{s}\right) C_{SO}\left(\frac{g}{g}\right)}{\sqrt{\pi\alpha\left(\frac{cm^2}{s}\right)t(s)}}$$

where $$\alpha = \frac{E\ D_{ei}}{E + P_s(1-E)\frac{K_d}{H}}$$

$$= \frac{D_{ei}}{1 + S \cdot K}$$

with $S = \frac{1-E}{E}$, and $K = \frac{K_d}{H} P_s$

$Dei = 0.05\ (0.35)^{1/3} = 0.0352\ cm^2/s$

$$\frac{H}{K_d} \text{ in } \frac{g\ soil}{cm^3\ air} = \frac{8.37 \times 10^{-3}}{1000} \times 41 = 0.000343\ \frac{mg/cm^3\ air}{mg/g\ soil}$$

Hence:

$$\alpha = \frac{0.0123}{0.35 + 2.65(0.65)\frac{1}{0.000343}} = 2.45 \times 10^{-6}\ \text{cm}^2/\text{s}$$

The average emission rate when the initial concentration in soil, $C_{S)} = 10^{-6}$ g/g (= 1 ppm) can be obtained by averaging over 10 days or 70 years:

- 10 day (= 86,400 sec) average:

$$\overline{N}_A = 0.00034(0.35)\ \frac{2(0.0352)(10^{-6})}{\sqrt{3.14(2.45\text{x}10^{-6})(864,000)}} = 3.25 \times 10^{-12}\ \text{g/cm}^2\cdot\text{s}$$

- 70 years (= 2.2075 x 10^9 sec) average:

$$\overline{N}_A = 0.00034(0.35)\ \frac{2(0.0352)(10^{-6})}{\sqrt{3.14(2.45\text{x}10^{-7})(2.2075\text{x}10^9)}} = 6.4 \times 10^{-14}\ \text{g/cm}^2\cdot\text{s}$$

Case 2. Unsteady-state emission--soil cover applied.

Clean soil is applied on top of contaminated soil surface. Emissions will occur through the cover material, and will remain unsteady below the vapor saturation point. The average emission rate under the assumption of the initial contamination depth, L = 200 cm, can be obtained by Eq. (A-13):

$$\overline{N}_A = \frac{2(H/K_d)C_{S0}\cdot E\cdot Dei}{L} \cdot \frac{1}{t}\sum_{n=0}^{\infty}\left[\int_0^t e^{-\frac{\alpha\,(2n+1)^2\Pi^2}{4L^2}t}\,dt\cdot\cos\left\{\frac{(2n+1)}{2}\frac{\Pi\ell}{L}\right\}\right]$$

For an average emission over a period of 10 days (864,000 sec)

$$\overline{N}_A = \frac{2(0.000342)(10^{-6})(0.35)(0.0352)}{(200)(864,000)}$$

$$\sum_{n=0}^{\infty}\int_0^{864,000} e^{-\frac{2.45\times10^{-6}(2n+1)^2}{4(200)^2}\Pi^2 t}\,dt\cdot\cos\left\{\frac{(2n+1)}{2}\frac{\Pi(25.4)}{200}\right\}$$

$= 7.6 \times 10^{-14}$ g/cm$^2\cdot$s

Similarly, the average emission rate over a period of 70 years

$= 5 \times 10^{-14}$ g/cm$^2\cdot$s

NOTE: The emission rate equation is programmed in a computer, and the summation is carried out using the program.

Case 3. Steady-state emission--no cover.

The same scenario assumes that the contaminated surface is exposed to the atmosphere, and its surface is maintained at the concentration of interest over the period of emission. This will apply to the case where a large reservoir of PCBs is available for emission until the concentration at the surface reaches the saturation value. This will be the upper-bound emission rate for contaminated soil with no clean cover applied.

PCB-1254

$$\frac{H}{K_d} \cdot C_s = \frac{0.34 \text{ g/cm}^3 \text{ air/g/cm}^3 \text{ water}}{1000 \text{ mg/g soil/mg/cm}^3 \text{ water}} C_s$$

$$= 0.00034 \frac{\text{mg/cm}^3 \text{ air}}{\text{mg/g soil}} \; 0.001 \text{ mg/g soil}$$

$$= 3.4 \times 10^{-7} \text{ mg/cm}^3 \text{ air} = 3.4 \times 10^{-7} \times 10^{-3} \times 10^{-6} \; \mu\text{g/m}^3$$

$$= 340 \; \mu\text{g/m}^3$$

Partitioned vapor concentration: 340 μg/m^3

Vapor pressure at 25°C = 7.71×10^{-5} mmHg

$$C^* = \frac{PMW}{RT} = \frac{14.7 \; (7.71 \times 10^{-5}/760) \cdot 328.4}{10.73 \; (460 + 77)} = 8.5 \times 10^{-8} \; \#/\text{ft}^3$$

where F = 1.8 (25) + 32 = 77°F,
C^* = saturation concentration of PCB-1254 in #/ft^3 or μg/m^3,
P = vapor pressure,
MW = molecular weight,
R = gas constant.

$$\frac{8.5 \times 10^{-8} \; (454) \times 10^6 \; \mu\text{g}}{(0.3048)^3 \text{ m}^3} = 1362.7 \; \mu\text{g/m}^3$$

When PCB-1254 is greater than 4 ppm in soil, the partial pressure in the air phase is equal to vapor pressure. An average temperature of 25°C is used for evaluating the saturation concentration in the vapor phase. Since the vapor pressure is dependent upon temperature, a specific evaluation will require a temperature of interest. The temperature in the subsurface soil may not fluctuate considerably over the average value.

PCB-1242

Concentration in air above soil with 1 μg/g PCB

$$340 \times \frac{5.73\text{x}10^{-4}}{8.37\text{x}10^{3}} = 23.3\ \mu\text{g/m}^3$$

Saturated vapor concentration

$$1362.7\ \frac{4.06\text{x}10^{-4}}{7.71\text{x}10^{-5}}\ \frac{266.5}{328.4} = 5823.3\ \mu\text{g/m}^3$$

Note: When the PCB concentration in soil = 250 μg/g = $\left(\frac{5823.3}{23.3}\right)$, the vapor phase is saturated.

In order to calculate the emission rate, the values for gas-phase mass transfer coefficients are estimated using the relationship given by Hwang (1982).

$$\text{PCB-1254; kg} = 5.8\text{x}10^{-5}\ \left(\frac{18}{328.4}\right)^{0.5} = 1.36\text{x}10^{-5}\ \text{g mol/cm}^2 \cdot \text{s}$$

$$\text{PCB-1242; kg} = 5.8\text{x}10^{-5}\ \left(\frac{18}{266.5}\right)^{0.5} = 1.51\text{x}10^{-5}\ \text{g mol/cm}^2 \cdot \text{s}$$

Mole fractions of PCBs in the gas phase (y) when the concentrations are retained at 340 μg/m^3 and 23.3 μg/m^3 for PCB-1254 and PCB-1242, respectively, are:

PCB-1254 $y = 340\ \mu g/m^3 \cdot 0.0742\ ppb/(\mu g/m^3) \times 10^{-9} = 2.52 \times 10^{-8}$

PCB-1242 $y = 23.3\ \mu g/m^3 \cdot 0.0916\ ppb/(\mu g.m^3) \times 10^{-9} = 2.13 \times 10^{-9}$

The emission rate, Q, can be obtained from the formula $Q = MW \cdot kg \cdot y$, where MW = molecular weight (Hwang, 1982). Thus, the emission rate for each Aroclor is:

PCB-1254 $Q = 328.4\ (1.36 \times 10^{-5})(2.52 \times 10^{-8}) = 1.13 \times 10^{-10}$ g/s cm^2

PCB-1242 $Q = 266.5\ (1.51 \times 10^{-5})\ (2.13 \times 10^{-9}) = 8.57 \times 10^{-12}$ g/s cm^2

Case 4. Steady-state emission--cover applied.

It is assumed that the contaminated site is covered with PCB-free soil cover, and that the concentration at the top of the contaminated soil is maintained at 1 μg/g over the period of emission. The emission rate can be obtained from the formula (Hwang, 1982).

$$Q = D_i\ {P_T}^{4/3}\ \frac{C_i^*}{h}\ ,\ g/cm^2 \cdot s$$

where D_i = diffusivity, cm^2/s,
P_T = total porosity,
C_i^* = true vapor pressure in equilibrium with soil, g/cm^3,
h = cover thickness, cm.

Using the diffusivity value of 0.05 cm^2/s, one can get for PCB-1254, an emission rate of

$$Q = (0.05)(0.35)^{4/3}\ \frac{340\ \mu g/m^3\ 10^{-6}\ g/\mu g \times 10^{-6}\ cm^3/m^3}{25.4\ cm}$$

$$= 1.67 \times 10^{-13}\ g/cm^2 \cdot s$$

Similarly, for PCB-1242:

$$Q = (0.05)\ (0.35)^{4/3}\ \frac{23.3\text{x}10^{-6}\ \text{x}\ 10^{-6}}{25.4} = 1.14\text{x}10^{-14}\ \text{g/cm}^2 \cdot \text{s}$$

REFERENCES (APPENDIX B)

Hwang, S.T. (1982) Toxic emissions from land disposal facilities. Environ. Prog. 1:46.

APPENDIX C

SUMMARY OF COMPUTER RUNS FOR EACH AROCLOR AND AT EACH VALUE OF SOIL-AIR PARTITION COEFFICIENTS

TABLE C-1. PERMISSIBLE PCB-1242 SOIL CONTAMINATION LEVELS[g] (UNCOVERED SURFACE CONTAMINATION, K_d = 1000)

Location and route of human exposure	Permissible levels (μg/g) corresponding to					
	Noncancer short-term[a] acceptable intake μg/day[b]		Cancer risk specific doses (μg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	55	510	0.008	0.08	0.8	8
- Soil ingestion[d], inhalation[e]	92	2100	0.06	0.6	6	61
- Inhalation only[e]	116	vs	0.2	2	20	204
0.1 km from contaminated site - Inhalation only[e]	vs[f]	vs	20	204	1.1×10^4	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	428	3.1×10^4	vs	vs

[a] Short-term ≡ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquid for the limit.

[g] Soil-air partition coefficient = 2.35×10^{-5} g soil/cm^3 air (= $H.41/K_d$ = 5.73×10^{-4} (41)/1000 = 2.35×10^{-5}).

TABLE C-2. PERMISSIBLE PCB-1242 SOIL CONTAMINATION LEVELS[g] (UNCOVERED SURFACE CONTAMINATION, $K_d = 40$)

Location and route of human exposure	Permissible levels (μg/g) corresponding to					
	Noncancer short-term[a] acceptable intake μg/day[b]		Cancer risk specific doses (μg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	60	690	.01	0.1	1.0	13
- Soil ingestion[d], inhalation[e]	247	2800	.03	0.3	3.0	35
- Inhalation only[e]	vs[f]	vs	0.04	.4	4	110
0.1 km from contaminated site - Inhalation only[e]	7	vs	4	110	1.1×10^4	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	310	3.1×10^4	vs	vs

[a] Short-term ≡ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquid for the limit.

[g] Soil-air partition coefficient = 2.35×10^{-5} g soil/cm^3 air (= $H.41/K_d = 5.73 \times 10^{-4}$ (41)/1000 = 2.35×10^{-5}).

TABLE C-3. PERMISSIBLE PCB-1248 SOIL CONTAMINATION LEVELS[g] (UNCOVERED SURFACE CONTAMINATION, K_d = 1000)

	Permissible levels (µg/g) corresponding to					
	Noncancer short-term[a] acceptable intake µg/day[b]		Cancer risk specific doses (µg/day)			
Location and route of human exposure	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	32	612	0.01	0.1	1	10
- Soil ingestion[d], inhalation[e]	42	2500	0.04	0.5	5	49
- Inhalation only[e]	47	vs	0.08	0.8	4	110
0.1 km from contaminated site - Inhalation only[e]	vs[f]	vs	8.0	110	8,700	8.7×10^5
1 km from contaminated site - Inhalation only[e]	vs	vs	270	2.5×10^4	vs	vs

[a] Short-term ≡ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquid for the limit.

[g] Soil-air partition coefficient = 2.35×10^{-5} g soil/cm^3 air (= $H.41/K_d$ = 5.73×10^{-4} (41)/1000 = 2.35×10^{-5}).

TABLE C-4. PERMISSIBLE PCB-1248 SOIL CONTAMINATION LEVELS[g]
(UNCOVERED SURFACE CONTAMINATION, K_d = 40)

Locatio and route of human exposure	Permissible levels (μg/g) corresponding to					
	Noncancer short-term[a] acceptable intake μg/day[b]		Cancer risk specific doses (μg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	80	710	0.01	0.1	1	8
- Soil ingestion[d], inhalation[e]	330	2900	0.02	0.2	2.0	37
- Inhalation only[e]	vs[f]	vs	0.02	0.2	2.0	87
0.1 km from contaminated site - Inhalation only[e]	vs	vs	2.0	90	8,700	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	250	2.5×10^4	vs	vs

[a] Short-term ≅ 10-day intake.
[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.
[c] Children ages 1-5, with pica (consuming 3 g soil/day).
[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).
[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.
[f] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquid for the limit.
[g] Soil-air partition coefficient = 2.35×10^{-5} g soil/cm^3 air (= $H.41/K_d = 5.73 \times 10^{-4}$ (41)/1000 = 2.35×10^{-5}).

TABLE C-5. PERMISSIBLE PCB-1254 SOIL CONTAMINATION LEVELS[g] (UNCOVERED SURFACE CONTAMINATION, K_d = 1000)

Location and route of human exposure	Permissible levels (μg/g) corresponding to					
	Noncancer short-term[a] acceptable intake μg/day[b]		Cancer risk specific doses (μg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	90	720	0.01	0.1	1	12
- Soil ingestion[d], inhalation[e]	370	2980	0.04	0.4	4	59
- Inhalation only[e]	vs[f]	vs	0.05	0.5	7	460
0.1 km from contaminated site - Inhalation only[e]	vs	vs	7	460	4.7×10^4	4.7×10^6
1 km from contaminated site - Inhalation only[e]	vs	vs	1.2×10^3	1.3×10^5	vs	vs

[a] Short-term ≡ 10-day intake.
[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.
[c] Children ages 1-5, with pica (consuming 3 g soil/day).
[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).
[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.
[f] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquid for the limit.
[g] Soil-air partition coefficient = 2.35×10^{-5} g soil/cm^3 air (= $H.41/K_d$ = 5.73×10^{-4} (41)/1000 = 2.35×10^{-5}).

TABLE C-6. PERMISSIBLE PCB-1254 SOIL CONTAMINATION LEVELS[g]
(UNCOVERED SURFACE CONTAMINATION, $K_d = 40$)

	Permissible levels (µg/g) corresponding to					
	Noncancer short-term[a] acceptable intake µg/day[b]		Cancer risk specific doses (µg/day)			
Location and route of human exposure	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	100	730	0.009	0.09	2.0	12
- Soil ingestion[d], inhalation[e]	420	3000	0.01	0.1	3	36
- Inhalation only[e]	vs[f]	vs	0.01	0.1	5	470
0.1 km from contaminated site - Inhalation only[e]	vs	vs	5	470	4.7×10^4	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	1300	1.3×10^5	vs	vs

[a] Short-term ≡ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquid for the limit.

[g] Soil-air partition coefficient = 2.35×10^{-5} g soil/cm^3 air (= $H.41/K_d = 5.73 \times 10^{-4}$ (41)/1000 = 2.35×10^{-5}).

TABLE C-7. PERMISSIBLE PCB-1260 SOIL CONTAMINATION LEVELS[g]
(UNCOVERED SURFACE CONTAMINATION, K_d = 1000)

Location and route of human exposure	Permissible levels (μg/g) corresponding to					
	Noncancer short-term[a] acceptable intake μg/day[b]		Cancer risk specific doses (μg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	25	640	0.01	0.1	1	12
- Soil ingestion[d], inhalation[e]	61	2670	0.04	0.4	4	48
- Inhalation only[e]	vs[f]	vs	0.06	0.6	6	91
0.1 km from contaminated site - Inhalation only[e]	vs	vs	6	91	7.7×10^3	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	240	2.2×10^4	vs	vs

[a] Short-term ≡ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquid for the limit.

[g] Soil-air partition coefficient = 2.35×10^{-5} g soil/cm^3 air (= $H.41/K_d = 5.73 \times 10^{-4}$ (41)/1000 = 2.35×10^{-5}).

TABLE C-8. PERMISSIBLE PCB-1260 SOIL CONTAMINATION LEVELS[g] (UNCOVERED SURFACE CONTAMINATION, K_d = 40)

Location and route of human exposure	Permissible levels (μg/g) corresponding to					
	Noncancer short-term[a] acceptable intake μg/day[b]		Cancer risk specific doses (μg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	87	710	0.01	0.1	1.0	17
- Soil ingestion[d], inhalation[e]	360	2900	0.01	0.1	1.0	40
- Inhalation only[e]	vs[f]	vs	0.01	0.1	1.0	77
0.1 km from contaminated site - Inhalation only[e]	vs	vs	1.0	76	7600	7.6×10^5
1 km from contaminated site - Inhalation only[e]	vs	vs	220	2.2×10^4	vs	vs

---

[a] Short-term ≡ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquid for the limit.

[g] Soil-air partition coefficient = 2.35×10^{-5} g soil/cm^3 air (= $H.41/K_d = 5.73 \times 10^{-4}\ (41)/1000 = 2.35 \times 10^{-5}$).

TABLE C-9. PERMISSIBLE PCB-1242 SOIL CONTAMINATION LEVELS[g]
(25-cm-THICK CLEAN SOIL COVER, K_d = 1000)

Location and route of human exposure	Permissible levels (μg/g) corresponding to					
	Noncancer short-term[a] acceptable intake μg/day[b]		Cancer risk specific doses (μg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	200	1400	0.2	2	17	170
- Soil ingestion[d], inhalation[e]	820	5700	0.6	6	48	260
- Inhalation only[e]	vs[f]	vs	0.9	9	86	vs
0.1 km from contaminated site - Inhalation only[e]	vs	vs	85	vs	vs	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	vs	vs	vs	vs

[a] Short-term ≅ 10-day intake.
[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.
[c] Children ages 1-5, with pica (consuming 3 g soil/day).
[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).
[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.
[f] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquid for the limit.
[g] Soil-air partition coefficient = 2.35×10^{-5} g soil/cm^3 air (= $H.41/K_d$ = 5.73×10^{-4} (41)/1000 = 2.35×10^{-5}).

TABLE C-10. PERMISSIBLE PCB-1242 SOIL CONTAMINATION LEVELS[g]

(25-cm-THICK CLEAN SOIL COVER, $K_d = 40$)

Location and route of human exposure	Permissible levels (µg/g) corresponding to					
	Noncancer short-term[a] acceptable intake µg/day[b]		Cancer risk specific doses (µg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	170	1200	0.03	0.3	3.0	vs
- Soil ingestion[d], inhalation[e]	450	3100	0.1	1.0	12	vs
- Inhalation only[e]	vs[f]	vs	1.0	vs	vs	vs
0.1 km from contaminated site - Inhalation only[e]	vs	vs	vs	vs	vs	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	vs	vs	vs	vs

[a] Short-term ≅ 10-day intake.
[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.
[c] Children ages 1-5, with pica (consuming 3 g soil/day).
[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).
[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.
[f] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquid for the limit.
[g] Soil-air partition coefficient = 2.35×10^{-5} g soil/cm^3 air (= $H.41/K_d = 5.73 \times 10^{-4}$ (41)/1000 = 2.35×10^{-5}).

TABLE C-11. PERMISSIBLE PCB-1248 SOIL CONTAMINATION LEVELS[g] (25-cm-THICK CLEAN SOIL COVER, K_d = 1000)

Location and route of human exposure	Permissible levels (μg/g) corresponding to					
	Noncancer short-term[a] acceptable intake μg/day[b]		Cancer risk specific doses (μg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	190	1300	0.09	1	10	26
- Soil ingestion[d], inhalation[e]	650	4500	0.1	1	10	93
- Inhalation only[e]	vs[f]	vs	0.1	1	14	19,000
0.1 km from contaminated site - Inhalation only[e]	vs	vs	14	19,000	vs	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	vs	vs	vs	vs

[a] Short-term ≡ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquid for the limit.

[g] Soil-air partition coefficient = 2.35×10^{-5} g soil/cm^3 air (= $H.41/K_d$ = 5.73×10^{-4} (41)/1000 = 2.35×10^{-5}).

TABLE C-12. PERMISSIBLE PCB-1248 SOIL CONTAMINATION LEVELS[g]
(25-cm-THICK CLEAN SOIL COVER, K_d = 40)

Location and route of human exposure	Permissible levels (μg/g) corresponding to					
	Noncancer short-term[a] acceptable intake μg/day[b]		Cancer risk specific doses (μg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	160	1100	0.01	0.1	1.0	460
- Soil ingestion[d], inhalation[e]	vs[f]	vs	0.02	0.2	2.0	2500
- Inhalation only[e]	vs	vs	0.02	0.2	2.0	1.9×10^{4}
0.1 km from contaminated site - Inhalation only[e]	vs	vs	2.0	1.9×10^{4}	vs	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	vs	vs	vs	vs

[a] Short-term ≡ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquid for the limit.

[g] Soil-air partition coefficient = 2.35×10^{-5} g soil/cm^3 air (= $H.41/K_d$ = 5.73×10^{-4} (41)/1000 = 2.35×10^{-5}).

TABLE C-13. PERMISSIBLE PCB-1254 SOIL CONTAMINATION LEVELS[g]
(25-cm-THICK CLEAN SOIL COVER, K_d = 1000)

Location and route of human exposure	Permissible levels (μg/g) corresponding to					
	Noncancer short-term[a] acceptable intake μg/day[b]		Cancer risk specific doses (μg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	180	1300	0.02	0.2	2	vs
- Soil ingestion[d], inhalation[e]	520	4000	0.06	0.6	8	vs
- Inhalation only[e]	vs[f]	vs	0.08	0.8	14	vs
0.1 km from contaminated site - Inhalation only[e]	vs	vs	14	vs	vs	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	vs	vs	vs	vs

[a] Short-term ≡ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquid for the limit.

[g] Soil-air partition coefficient = 2.35×10^{-5} g soil/cm^3 air (= $H.41/K_d$ = 5.73×10^{-4} (41)/1000 = 2.35×10^{-5}).

TABLE C-14. PERMISSIBLE PCB-1254 SOIL CONTAMINATION LEVELS[g]
(25-cm-THICK CLEAN SOIL COVER, $K_d = 40$)

Location and route of human exposure	Permissible levels (µg/g) corresponding to					
	Noncancer short-term[a] acceptable intake µg/day[b]		Cancer risk specific doses (µg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	140	970	0.01	0.1	7	vs
- Soil ingestion[d], inhalation[e]	vs	vs	0.02	0.2	9	vs
- Inhalation only[e]	vs	vs	0.02	0.2	10	vs
0.1 km from contaminated site - Inhalation only[e]	vs	vs	10	vs	vs	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	vs	vs	vs	vs

[a] Short-term ≡ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquid for the limit.

[g] Soil-air partition coefficient = 2.35×10^{-5} g soil/cm^3 air (= $H.41/K_d = 5.73 \times 10^{-4}$ (41)/1000 = 2.35×10^{-5}).

TABLE C-15. PERMISSIBLE PCB-1260 SOIL CONTAMINATION LEVELS[g]
(25-cm-THICK CLEAN SOIL COVER, K_d = 1000)

Location and route of human exposure	Permissible levels (µg/g) corresponding to					
	Noncancer short-term[a] acceptable intake µg/day[b]		Cancer risk specific doses (µg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	184	1300	0.02	0.2	2.0	22
- Soil ingestion[d], inhalation[e]	550	4000	0.07	0.7	7	120
- Inhalation only[e]	vs[f]	vs	0.08	0.8	8.0	770
0.1 km from contaminated site - Inhalation only[e]	vs	vs	8.0	770	vs	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	vs	vs	vs	vs

[a] Short-term ≡ 10-day intake.

[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.

[c] Children ages 1-5, with pica (consuming 3 g soil/day).

[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).

[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.

[f] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquid for the limit.

[g] Soil-air partition coefficient = 2.35×10^{-5} g soil/cm^3 air (= $H.41/K_d$ = 5.73×10^{-4} (41)/1000 = 2.35×10^{-5}).

TABLE C-16. PERMISSIBLE PCB-1260 SOIL CONTAMINATION LEVELS[g]
(25-cm-THICK CLEAN SOIL COVER, K_d = 40)

Location and route of human exposure	Permissible levels (μg/g) corresponding to					
	Noncancer short-term[a] acceptable intake μg/day[b]		Cancer risk specific doses (μg/day)			
	100 for child	700 for adult	0.00175 (10^{-7} risk)	0.0175 (10^{-6} risk)	0.175 (10^{-5} risk)	1.75 (10^{-4} risk)
On the contaminated site						
- Soil ingestion[c], inhalation[e]	110	800	0.01	0.1	1.0	360
- Soil ingestion[d], inhalation[e]	800	5000	0.02	0.2	1.0	550
- Inhalation only[e]	vs[f]	vs	0.02	0.2	1.0	620
0.1 km from contaminated site - Inhalation only[e]	vs	vs	1.0	620	vs	vs
1 km from contaminated site - Inhalation only[e]	vs	vs	vs	vs	vs	vs

[a] Short-term ≅ 10-day intake.
[b] Based on average weights of 10 and 70 kg for a child and an adult, respectively.
[c] Children ages 1-5, with pica (consuming 3 g soil/day).
[d] Children ages 1-5, without pica (consuming 0.6 g soil/day).
[e] Inhalation rates are assumed to be 20 m^3/day for the short-term and longer-term noncancer exposures; all other (more chronic) exposures assumed to be 10 m^3/day as a result of 182 days exposure per year.
[f] vs denotes no theoretical upper-bound limit. Practical reasons require no free-flowing PCB liquid for the limit.
[g] Soil-air partition coefficient = 2.35×10^{-5} g soil/cm^3 air (= $H.41/K_d = 5.73 \times 10^{-4}$ (41)/1000 = 2.35×10^{-5}).

APPENDIX D

HEALTH ADVISORIES FOR PCBs IN SOIL

Prepared by

Michael L. Dourson
Environmental Criteria and Assessment Office
Office of Health and Environmental Assessment
U.S. Environmental Protection Agency
Cincinnati, Ohio

APPENDIX D

CONTENTS

EXISTING GUIDELINES, RECOMMENDATIONS, AND STANDARDS D-2

NONCARCINOGENIC EFFECTS D-4

QUANTIFICATION OF SHORT-TERM HEALTH ADVISORY LEVELS D-11

CARCINOGENIC EFFECTS. D-14

QUANTIFICATION OF CARCINOGENIC RISK D-17

SPECIAL CONSIDERATIONS. D-20

  High-Risk Subpopulation. D-20
  Cocarcinogenesis, Initiation, and Promotion. D-21

SUMMARY D-21

REFERENCES. D-25

EXISTING GUIDELINES, RECOMMENDATIONS, AND STANDARDS

The manufacture, sale, and distribution of PCBs have been restricted under Section 6(e) of the Toxic Substances Control Act (TSCA) (P.L. 94-469). PCBs were restricted to sealed systems as of 1977, and manufacture and distribution were banned in 1979. Rules for the disposal of PCBs were proposed in 1978 (43 FR 7150).

The U.S. EPA (1980) has set ambient water quality criteria for PCBs for the protection of humans from increased risk of cancer over a lifetime of 10^{-5}, 10^{-6} and 10^{-7} at 0.79, 0.079, and 0.0079 ng/L. As a result of the large bioconcentration factor in fish, these criteria apply regardless of whether exposure occurs through consumption of 2 L of water and 6.5 g of fish/day or through consumption of fish alone. The Food and Drug Administration (FDA) has set temporary tolerances for PCBs in food and food related products, as shown in Table D-1.

Occupational exposure limits for PCBs have been recommended by the American Conference of Governmental Industrial Hygienists (ACGIH, 1980), and criteria have been set for PCBs in workplace air by the National Institute for Occupational Safety and Health (NIOSH, 1977). The time-weighted average (TWA) and short-term exposure limit (STEL) for Aroclor 1254, are, respectively, 0.5 and 1.0 mg/m^3, and for Aroclor 1242, 1 and 2 mg/m^3 (ACGIH, 1980). The NIOSH (1977) criterion is 1.0 µg/m^3 for a 10 hours/day, 40 hours/week exposure.

The National Academy of Sciences developed a 24-hour suggested no adverse response level (SNARL) for PCBs of 350 µg/L based on the induction of mixed-function oxidase enzymes in the liver of rats administered Aroclor at doses of 1 to 2 mg/kg (NAS, 1980). For this analysis, an uncertainty factor of 100 was used, since only enzyme induction was reported in this dose range.

TABLE D-1. FDA REGULATIONS FOR PCBs

Commodity	Temporary tolerances (ppm)
Milk (fat basis)	1.5
Manufactured dairy products (fat basis)	1.5
Poultry (fat basis)	3.0
Eggs	0.3
Finished animal feeds	0.2
Animal feed components of animal origin	2.0
Edible portion of fish and shellfish	5.0
Infant and junior foods	0.2
Paper food packaging material	10.0

SOURCE: 21 CFR 109.30.

NONCARCINOGENIC EFFECTS

Tests of the acute lethality of PCB products in laboratory animals, with the exception of the guinea pig, suggest that, in general, PCB products have similar toxicity regardless of route of administration, species, or age of animal. The single dose oral LD_{50} value in rats, rabbits, mice, and mink ranged from 0.5-20 g/kg bw (Grant and Phillips, 1974; Bruckner et al., 1973; Kimbrough et al., 1978; Fishbein, 1974; Garthoff et al., 1981, Aulerich and Ringer, 1977). Route of administration also had little effect (less than one order of magnitude) on lethality, with the lethal dose for dermal administration in rabbits ranging from 0.7-3 g/kg bw (Nelson et al., 1972), while the lethal dose in mice administered PCBs by intraperitoneal injection ranged from 0.8-1.2 g/kg bw (Lewin et al., 1972).

There are only two indications of major differences in the acute toxicity of PCBs. First, there is limited evidence that the guinea pig may be more sensitive to the lethality of PCBs than other species. Miller (1944) observed a 100% mortality in a small group of guinea pigs receiving two oral doses of PCB (43% chlorine) at levels of 67 mg/animal at an interval of 7 days apart; and McConnell and Kinney (1978) reported an LD_{50-30} of 0.5 mg/kg for the PCB isomer 3,4,5-sym-hexachlorobiphenyl (HCB) in guinea pigs. This indication of possible large interspecies differences in sensitivity is of concern in species-to-species extrapolation when there is insufficient data to indicate which experimental animal most accurately reflects the sensitivity of humans. The second problem concerns the possible large difference in toxicity of specific isomers of PCBs. There are indications, on the basis of limited data available from Biocca et al. (1981), that four different hexa-PCBs differ in LD_{50} value from 19 mg/kg to >64 mg/kg after oral administration to mice. It would not be unreasonable to assume that even larger differences will be encountered as more isomers are tested. Variation in toxicity of the different isomers is not of great concern in defining acceptable environmental levels, since individual isomers were not commercially made and released to the environment. The analytical methods used to measure environmental PCBs determine the levels, in mg, of specific Aroclors. Since, as described above, the Aroclors do not differ greatly in acute toxicity, using data from the most toxic Aroclor should be protective without overly penalizing the other Aroclors.

Some data are available on the nonlethal acute toxicity of PCBs administered by the oral route for periods of 30 days or less. The effects described in these studies were alterations of the liver, thyroid, and reproductive system. Rosin and Martin (1983) reported that a dose of 500 mg/kg of Aroclor 1254 for 14 days to CD-1 mice decreased pentobarbital sleeping time, indicating a substantial induction of microsomal enzymes. At lower doses, Sanders et al. (1974) reported that exposure of ICR mice to diets containing 250 to 62.5 ppm of Aroclor 1254 for 14 days resulted, respectively, in hepatomegaly and elevated serum corticosterone (the latter presumably as a result of altered liver steroid metabolism). These exposures would result in doses of 32.8 and 8.1 mg/kg bw/day, assuming that a mouse consumes 13% of its body weight per day.

Similarly, Narbonne (1979) reported decreases in phenobarbital sleeping time in Sprague-Dawley rats maintained fro 8 days on a diet containing 100 ppm of Phenoclor DP6 (dose of 5 mg/kg bw/day, assuming that a rat consumes 5% of its body weight/day). PCB-induced increases in liver enzymes were suggested as the reason for the increase in testicular acid phosphatase observed by Dikshith et al. (1975) in Sprague-Dawley rats fed Aroclor 1254 at a dose of 50 mg/kg bw/day for 7 days. Increases in liver-to-body weight ratio appear to be one of the sensitive indicators of PCB exposure. Carter and Mercer (1983) reported that 64 mg/kg bw/day of Aroclor 1254 caused increased liver weight, while Grant and Phillips (1974) observed increased liver weight at doses as low as 12.5 and 5 mg/kg bw/day in male and female Wistar rats, respectively, receiving Aroclor 1254 in the diet for 14 days. Carter (1983) observed hepatomegaly in rats ingesting diets containing as little as 20 ppm Aroclor 1254 (1 mg/kg bw/day) for 14 days. Doses as low as 1 ppm in diet of 3,4,5,3',4',5'-hexachlorobiphenyl (345 HCB) for 28 days caused liver microabscesses and an increased liver weight in 18-20 g 5-week-old C57Bl/6J mice (Biocca et al., 1981). Although adverse this study, 0.3 ppm in diet could be considered a lowest observed adverse effects were observed at concentrations as low as 0.3 ppm in diet in this study, there is no documentation indicating that commercial Aroclors contain 345 HcB. Hence, this study for 345 HcB cannot be used for establishing the short-term Health Advisory (HA), corresponding to an acceptable intake (AI) level.

Besides changes in the liver, other effects reported for exposure to low levels of PCBs were increased thyroid activity in Sherman rats maintained on diets containing 250 ppm of Aroclor 1254 (12.5 mg/kg bw) for 14 days; in Sprague-Dawley rats, administration of Aroclor 1254 by gavage for 21 days at a dose of 0.05 g/kg bw/day resulted in weight loss and decreased body temperature (Komives, 1979; Komives and Alayoku, 1980). Enlarged thyroid has also been found in Osborne-Mendel rats maintained on diets containing 5 ppm of Aroclor 1254 (0.25 mg/kg bw/day) for 4 weeks (Collins and Capen, 1980b). This exposure level also resulted in increased liver enzymes in Holtzman rats (Garthoff et al., 1977).

The toxicity of PCBs resulting from exposures of between 30 and 90 days has been more extensively studied. Alterations in liver histopathology occurred at doses as low as 5 ppm in diet for 5 weeks in Holtzman rats (Kasza et al., 1978b). In the mouse (MNRI), a dose of Clophen A-60, as low as 0.025 mg/mouse (0.8 mg/kg bw/day, assuming a mouse weight of 0.03 kg) for 62 days, increased the estrous cycle, probably as a result of PCB-induced changes in liver steroid metabolism (Orberg and Kihlstrom, 1973). At higher dietary concentrations of 167 ppm (22 mg/kg bw) for 6 weeks, Aroclor 1016 and 1242 decreased the immunologic capabilities of BALB/CJ mice (Loose et al., 1978).

Species other than rats and mice have been tested to a lesser extent for this duration. Rabbits exposed to diets containing 3 ppm of Aroclor 1254 (0.15 mg/kg bw/day, assuming that a rabbit consumes 4.9% of its body weight per day) for 8 weeks developed hepatomegaly and immunosuppression. In the guinea pig, Vos and van Genderen (1973) reported that diets containing 250 ppm of Clophen A-60 (7 mg/kg bw/day, assuming that a guinea pig consumes 2.8% of its body weight per day) for 4-7 weeks was lethal, while diets containing 50 ppm of Clophen A-60 or Aroclor 1260 (1.4 mg/kg bw) for 4 to 7 weeks produced immunosuppression. Allen et al. (1974) and Allen (1975) observed comedones and facial edema in rhesus monkeys ingesting diets containing 25 of ppm Aroclor (1.1 mg/kg bw, assuming that a monkey consumes 4.2% of its body weight per day) for 2 months.

Studies of subchronic and chronic exposure (i.e., $\geq$90 days) to PCBs have failed to use sufficiently low doses to define a no observed adverse effect level (NOAEL) in rats. In Sprague-Dawley rats, Allen et al. (1976) and Allen and Abrahamson (1979) reported that a 52-week exposure to diets containing 100 ppm of Aroclor 1248, 1254, or 1262 (5 mg/kg bw/day) followed by a 13-week observation period resulted in hepatomegaly and liver necrosis. At a lower exposure of 75 ppm in the diet (3.75 mg/kg bw/day) for 36 weeks, Sprague-Dawley rats developed focal necrosis (Jonsson et al., 1981). Elevated liver porphyrine levels were detected by both Kimbrough et al. (1972) and Zinkl (1977) after exposure of Sherman rats for 8 months to 20 ppm of Aroclor 1254 or 1260 (1 mg/kg bw/day) and of CD rats for 20 weeks to 10 ppm of Aroclor 1254 (0.4 mg/kg bw/day). In a study employing near-lifetime exposure (2 years), Morgan et al. (1981) reported an increase in mortality (33% as compared with 8% in controls) in Fischer 344 rats at the lowest dose tested (25 ppm; 1.25 mg/kg bw/day). The subchronic studies demonstrated increasing liver pathology over the dose ranges studied, 0.5-5 mg/kg bw/day; while in the only chronic study, the lowest dose tested (1.25 mg/kg bw/day) resulted in early deaths.

In mice, dietary exposure levels to Kanechlor-300, -400 or -500 or Arochlor 1254 of between 100 and 500 ppm (13-65 mg/kg bw/day) for periods from 23 weeks to 11 months produced hepatomegaly (Ito et al., 1973; Bell, 1983; Kimbrough and Linder, 1974). Koller (1977) used groups of BALB/CJ mice which were maintained for 9 months on diets containing 0, 3.75, 37.5, or 375 ppm of the Aroclors 121, 1242, or 1254 (0.46, 4.57, or 47.75 mg/kg bw/day). The Aroclor with the lowest chlorine content (1221) produced no liver lesions, while exposure to Aroclor 1242 resulted in increased liver weight in the high-dose group. In mice exposed to Aroclor 1254, increased mortality was observed in the high-dose group, mild hepatopathology was observed in the median dose group, and no liver lesions were detected in the low-dose group. The no observed effect level (NOEL) in this study in mice of 0.45 mg/kg bw/day is nearly identical to the lowest observed effect level (LOEL) of 0.5 mg/kg bw/ day associated with porphyria in rats (Kimbrough et al., 1972; Zinkl, 1977).

The only other species tested in chronic bioassays was the monkey, and it proved to be highly sensitive to the toxic effects of PCBs. The most common observations in monkeys exposed to Aroclor 1248 in the diet for a period of from 8 to 39 months were skin lesions, palpebral edema, and erythema (Barsotti and Allen, 1975; Allen and Barsotti, 1976; Allen et al., 1980; Becker et al., 1979). These effects were observed at the lowest doses tested, ranging from 2.5 to 3 ppm in the diet (0.095-0.126 mg/kg bw/day). In addition, Becker et al. (1979) reported that monkeys fed diets containing 3 ppm of PCBs had gastric lesions, body weight loss, and reduced hemoglobin and leukocytes. In the monkey, doses as low as 0.1 mg/kg bw/day produced frank toxic effects; no studies have been conducted from which a NOAEL can be derived or to indicate how close 0.1 mg/kg bw/day is to the NOAEL for monkeys.

Although PCBs have not been demonstrated to be animal teratogens following oral exposure, these compounds have been demonstrated to adversely affect reproduction. When adminstered to pregnant Wistar rats at a dose of 100 mg/kg bw/day on days 6 to 15 of gestation, Villeneuve et al. (1971) observed no adverse effects; however, using the same treatment schedule, Spencer (1982) reported that Sprague-Dawley rats were infertile after receiving 15 mg/kg bw/day, that animals receiving 5 mg/kg bw/day had reduced litter weights, and that 2.5 mg/kg bw/day was the NOEL. Rabbits had resorptions, abortions, and fetuses at similar dose levels of 12.5 mg/kg bw/day administered on days 0 to 28 of gestation; however, slightly smaller doses of 10 mg/kg bw/day were reported to be the NOEL. The Hartly guinea pig, which has been shown to have greater sensitivity to the toxicity of PCBs than most other species, had macerated fetuses after receiving 2.2 mg/day (6/5 mg/kg bw/day) of Clophen A-50 on days 10 to 60 of gestation (Brunstroem et al., 1982).

Effective doses of PCBs were lower than exposure occurred before and during gestation. In a two-generation study, Sherman rats maintained on diets containing 20 ppm Aroclor 1254 (1 mg/kg bw/day) had reduced litter size, and at 100 ppm (5 mg/kg bw/day) the pups that were born died during nursing (Linder et al., 1974). In this study, 5 ppm (0.25 mg/kg bw/day) was the NOEL. Complete loss of fertility was observed in male and female Wistar rats caged together for 9 weeks while ingesting 6.4 mg/kg bw/day of Aroclor 1254 emulsified in their drinking water (Baker et al., 1977). Males regained normal fertility after removal from treatment for 2 weeks. When Aroclor 1254 was administered to lactating Holtzman rats at 32 mg/kg bw/day on days 3, 5 and 7 of lactation, the future mating behavior of nursing male pups was adversely affected (Sager, 1983). A lower dose of 8 mg/kg bw/day was a NOEL.

Of the species tested, the mink and the monkey are the most sensitive to the reproductive toxicity of PCBs. Bleavins et al. (1980) maintained mink on diets containing 5 ppm Aroclor 1242 or 20 ppm Aroclor 1016 (doses of 0.75 and 3 mg/kg bw/day, assuming that a mink consumes 15% of its body weight per day) for 18 months and observed complete reproductive failure in the Aroclor 1242 group and 25% mortality and infertility in the Aroclor 1016 group. A more recent study by Aulerich et al. (1985) tested yet lower doses fed to mink via diet. Aroclor 1254 at 2.5 ppm; 3,4,5,3',4',5'-hexachlorobiphenyl (345 HCB) at 0.1 or 0.5 ppm; 2,4,5,2',4',5'-hexachlorobiphenyl (245 HCB) at 2.5 or 5.0 ppm; or 2,3,6,2',3',6'-hexachlorobiphenyl (236 HCB) at 2.5 or 5.0 ppm of diet were fed to groups of 10 standard dark mink (proven breeders). A group of 20 animals served as controls. All of the mink fed 0.5 ppm 345 HCB died within 60 days, while those fed 0.1 ppm showed 50% mortality after 3 months. One stillborn kit was whelped in the Aroclor 1254 group. 245 HCB and 236 HCB did not affect reproductive performance at either dose.

In a limited study (8 animals/group), Allen et al. (1980) maintained rhesus monkeys on diets containing 2.5 or 5 ppm (0.1 or 0.2 mg/kg bw/day) of Aroclor 1248 for 18 months. In the low-dose group, increased abortions were observed, while in the high-dose group, the mothers showed overt signs of toxicity and no live births occurred. After removal from exposure for 1 year, fertility had still not returned to normal, and some pups died during nursing. It is apparent that frank effects in reproduction were observed in mink at lower doses than in monkeys and still lower than the NOEL in rats, rabbits, and guinea pigs following repeated exposure to PCBs.

QUANTIFICATION OF SHORT-TERM HEALTH ADVISORY LEVELS

PCBs belong to a class of chemically stable, multi-use industrial chemicals that have been widely distributed in the ecosystem. Technical preparations consist of complex mixtures of discrete PCB isomers. Because of their physicochemical properties, PCBs have been used as heat exchangers, dielectric, hydraulic and lubricating fluids, plasticizers, pesticide extenders, adhesives, printing inks and surface coatings.

The physical and chemical properties and the chemical formulations of PCBs vary considerably, depending on the amount and position of chlorine substitution. Such properties as stability, volatility, and water solubility are particularly important in regard to frequency of occurrence in the environment. The higher chlorinated biphenyls are less volatile than the lower chlorinated biphenyls (Mieure et al., 1976). PCBs are extremely insoluble in water. The solubility of commercial mixtures (for example, the Aroclors) ranges from 25 to 200 ppb (25 to 200 μg/L), depending on the chlorine content (Nisbet and Sarofim, 1972; Haque et al., 1974). The solubility of discrete PCB isomers has been examined, and ranges from 1 to 600 ppb (1 to 600 μg/L) depending on the degree of chlorine substitution in the biphenyl ring (Haque and Schmeddig, 1975).

PCBs elicit a variety of adverse health effects. This is true of even partially well-defined compositions such as Aroclor 1254. For example, toxicology studies on Aroclor 1254 of less less than 30 days' duration report liver toxicity (Grant and Phillips, 1974; Carter, 1983), thyroid toxicity (Collins and Capen, 1980a, b, c) and reproductive toxicity (Villeneuve et al., 1971), as well as other types of toxicity (U.S. EPA, 1985b). At first, it would seem that this variety in elicited adverse effects would make it difficult to distinguish the critical toxic effects of PCBs. However, it appears that the experimental thresholds for these effects may be similar, at least for studies of 30 days' duration or less.

Villeneuve et al. (1971) found increased incidences of fetal death, resorptions, and abortions at 12.5 mg/kg/day of Aroclor 1254 in rabbits when exposed on days 1 through 28 of pregnancy. A dose of 1.0 mg/kg/day appeared to be without effect. Collins and Capen (1980a, b, c), in a series of studies on thyroid effecs in rats, determined that 50 ppm of diet (~ 2.5 to 5.0 mg/kg/day) for 4 weeks was associated with clearly defined adverse effects, but that doses of 5 ppm of diet (~ 0.25 to 0.5 mg/kg/day) were not. Carter (1983) demonstrated liver hepatomegaly in rats at doses of 20 ppm Aroclor 1254 of diet (~ 2 mg/kg/day) for 14 days; such an effect in the absence of toxicity (e.g., fatty infiltration of the liver) might not be considered adverse. Grant and Phillips (1974) observed increased liver weights at doses as low as 5 mg/kg/day Aroclor 1254 given in corn oil for 7 consecutive days. Collectively, these studies indicate that the experimental threshold for adverse effects of Aroclor 1254 in studies of 30 days' duration or less is at or near a dose of 1 mg/kg/day. Thus, it seems reasonable to use this latter dose as a basis for health risk assessments for Aroclor 1254 for short durations.

Utilizing a dose of 1 mg/kg/day weight as a No Adverse Effect dose, a 10-day exposure level for PCBs may be calculated as follows:

$$\text{10-day exposure level} = \frac{1\ \text{mg/kg/day} \times 10\ \text{kg}}{100} = 0.1\ \text{mg/day for a 10-kg child.}$$

where

10 kg = assumed body weight of a child, and

100 = uncertainty (safety) factor chosen in accordance with the National Academy of Sciences guidelines, in which a NOAEL from an animal study is employed.

For a 70-kg adult the 10-day exposure level would be 0.7 mg/day.

CARCINOGENIC EFFECTS

A number of short-term assays predictive of carcinogenic potential have been performed using the Aroclors and individual isomers of PCBs. Negative results have been obtained in the reverse mutation assay using *S. typhimurium* (Schoeny et al., 1979; Schoeny, 1982; Heddle and Bruce, 1977), and in the dominant lethal assay in rats (Green et al., 1975). PCB products also did not produce chromosomal changes in *D. melanogaster* (Nilsson and Ramel, 1974) or in the sperm and bone marrow cells of rats (Green et al., 1975; Garthoff et al., 1977; Dikshith et al., 1975). Wyndham et al. (1976), however, observed increases in reversion frequency in *S. typhimurium* exposed to 4-chlorobiphenyl and to a lesser extent with Aroclor 1221, while the more highly chlorinated 2,2',5,5'-tetrachlorobiphenyl and Aroclor 1254 were negative. The positive response was observed in one strain, TA1538, in the presence of a metabolic activation system derived from rabbit liver. In addition, a weak positive response was observed by Peakall et al. (1972) in an assay of chromosomal aberration in the embryos of ring doves fed Aroclor 1254. The variable data observed with PCBs is consistent with the poor response and lack of correlation with animal carcinogenicity data reported for many highly chlorinated compounds in short-term assay.

Early bioassays of PCBs were inadequate as a result of small group size or periods of exposure extending for less than 1 year. These studies failed to demonstrate that PCBs were carcinogenic when fed to rats or mice (Kimura and Baba, 1973; Ito et al., 1973; Ito et al., 1974; Kimbrough and Linder, 1974). The study by Kimbrough and Linder (1974) in which 50 BALB/CJ male mice were fed diets containing 300 ppm of Aroclor 1254 for 6 or 11 months was suggestive that Aroclor 1254 was a liver carcinogen. Of the 22 animals surviving PCB treatment for 11 months, all had areas of adenofibrosis in the liver, and seven had histologically identified hepatomas. In animals surviving 11 months which are maintained on PCB-contaminated diets for 6 months, and in control animals, there were, respectively, only 0/24 and 0/58 livers with areas of adenofibrosis and 1/24 and 0/58 animals with hepatomas. There were no histologically identified hepatocellular carcinomas in any group.

In a later study in rats using a larger group size and a longer exposure period, Kimbrough et al. (1975) reported an increased incidence in hepatocellular carcinomas in animals exposed to PCBs. In this study, 200 female Sherman rats were exposed to diets containing a nominal 100 ppm (range 70-107 ppm) of Aroclor 1260 for 630 days. The incidence of hepatocellular carcinomas in the treated group compared with control animals was 26/184 and 1/173, respectively; while the incidence of hepatic neoplastic nodules was 144/184 and 0/173, respectively.

In the only other chronic bioassay performed (NCI, 1978), 24 male and 24 female Fischer 344 rats/group were maintained on diets containing 0, 25, 50, or 100 ppm of Aroclor 1254. Although dose-related increases in nodular hyperplasia were observed, there was no significant increase in neoplastic lesions.

With respect to the tumor incidence reported by Kimbrough et al. (1975), the number of rats/group in the NCI study may have been too small to detect a carcinogenic response.

Very limited information is available on the carcinogenicity of PCBs in humans. In a survey of 1,200 patients in Yusho, Japan, 5.5 years after exposure to PCBs, 41% of the 22 deaths were attributed to neoplasia (Kuratsune, 1976; Urabe, 1974). The relevance of these findings is unclear since the tumors were at various common sites, and comparable incidences for an unexposed population were not presented. In two letters to the editor, Bahn et al. (1976, 1977) described suspected increases in malignant melanomas in a small group of workers exposed to Aroclor 1254. In the 31 "heavily exposed" workers, there were two cases of melanoma, while in the 41 "less heavily" exposed workers, there was one melanoma. The International Agency for Research on Cancer (IARC) estimates that only 0.04 cases would be expected in this number of individuals (IARC, 1978). The only epidemiologic study (Davidorf and Knupp, 1979) examined the association between ocular melanomas and populations residing in areas of known high environmental levels of PCBs. The authors concluded that a causal relationship between PCBs and ocular melanomas was not demonstrated. The IARC (1978) considered the association between exposure to Aroclor 1254 and malignant melanomas described in the two letters to the editor of the New England Journal of Medicine (Bahn et al., 1976, 1977) to be suggestive evidence that PCBs are human carcinogens.

QUANTIFICATION OF CARCINOGENIC RISK

WEIGHT OF EVIDENCE FOR HUMAN CARCINOGENICITY

The likelihood that a chemical such as PCB is a human carcinogen is expressed through a characterization or stratification of the "weight of evidence" (human, animal, short-term test) and a final indiction of the "overall weight of evidence" for human carcinogenicity.

The IARC has characterized the evidence for the carcinogenicity of PCBs in humans as "inadequate," the evidence for carcinogenicity in animals as "sufficient," and the supportive evidence from short-term tests as "inadequate," (IARC, 1982). The overall weight-of-evidence designation for PCBs under the IARC scheme is 2B (probably carcinogenic in humans; evidence inadequate in humans and sufficient in animals). The EPA has recently proposed a similar scheme, which is an adaptation of the IARC scheme (U.S. EPA, 1984a). There are some differences in the two schemes; however, for PCBs the requirements for inadequate evidence of carcinogenicity in humans and for sufficient evidence in animals are essentially identical. Similarly, the overall weight-of-evidence under the new EPA scheme would be designated as B2, which has the same requirements as the 2B designation of the IARC scheme.

To comply with the EPA proposed guidelines for carcinogen risk assessment (U.S. EPA, 1984a), any final risk estimate developed or used in risk characterization should be coupled with the EPA classification of the qualitative weight-of-evidence. Thus, those risk levels used in this paper are understood to carry the "B2" designation; for example, 1×10^{-5} (B2), 1×10^{-6} (B2), etc.

POTENCY SLOPE FACTORS

The use of a potency slope factor, necessary in calculating risk or back-calculating permissible PCB soil concentrations from selected risk levels, does not definitely indicate that the chemical is a human carcinogen. The likelihood that the agent is a human carcinogen is a function of the weight-of-evidence described above. The proposed EPA guidelines for carcinogen risk assessment (U.S. EPA, 1984a) suggest that agents falling into Groups A and B are suitable for quantitative risk assessments.

The U.S. EPA Carcinogen Assessment Group (CAG) has used the data from female rats in the study by Kimbrough et al. (1975) to quantify the carcinogenic risk from exposure to PCBs (U.S. EPA, 1980). In this analysis, the TWA concentration of PCB (Aroclor 1260) in the diet was determined to be 88.4 ppm, associated with a daily dose of 4.42 mg/kg bw/day, by assuming that an adult rat consumes food equal to 5% of its body weight per day. In addition, for this analysis, the incidence of hepatocellular carcinomas (26/184 in treated animals and 1/173 in controls) and neoplastic nodules (144/184 in treated animals and 0/173 in controls) were combined to produce tumor incidences of 170/184 and 1/173 in the treated and control groups, respectively. Using these data and the linearized multistage model, a cancer potency value (q_1^*) for human exposure to Aroclor 1260 of 4.34 $(mg/kg/day)^{-1}$ was calculated using the data in Table D-2. The U.S. EPA Office of Toxic Substances (OTS) has also used the data from the same study, but by altering two of the variables (see Table D-2), calculated a q_1^* of 3.57 $(mg/kg/day)^{-1}$ (U.S. EPA, 1985a). The average of these two values is 4.0 $(mg/kg/day)^{-1}$, and this value has been adopted by this health advisories appendix for use in developing advisory levels for PCBs clean-up.

Using this q_1^*, a risk-specific dose (RSD) of Aroclor 1260 that would result in an increased lifetime risk level of 10^{-5} for a 70-kg man can be calculated as follows:

TABLE D-2. DATA USED AS THE BASIS FOR THE q_1^*

Species	rat
Strain	Sherman
Sex	female
Body weight (assumed)	0.4 kg (0.35 kg)[a]
Length of exposure	645 days (730 days)[a]
Length of experiment	730 days
Tumor site	liver
Tumor type	combined hepatocellular carcinomas and neoplastic nodules
PCB product tested	Aroclor 1260

Dose (mg/kg/day)	Incidence (No. responding/No. tested)
0	1/173
4.42	170/184

---

[a] The data in parentheses indicate the alternative values used by OTS in computing a cancer potency factor.

SOURCE: Kimbrough et al., 1975.

$$\text{RSD} = 1 \times 10^{-5} \times 70 \text{ kg bw} \div q_1^* \text{ (mg/kg/day)}^{-1}$$
$$= 0.175 \ \mu\text{g/day}.$$

Thus, using the q_1^* for humans of 4.0 $(\text{mg/kg/day})^{-1}$, the RSDs corresponding to lifetime risks of 10^{-4}, 10^{-5}, and 10^{-6} are 1.75, 0.175, and 0.0175 µg/day, respectively. The adoption of these potency and concentration levels for all PCBs requires a further assumption that all PCB compounds are carcinogenic and that the potency of Aroclor 1260 is representative of any mixture of any other PCB compound.

SPECIAL CONSIDERATIONS

HIGH-RISK SUBPOPULATION

Two separate groups of high-risk subpopulations for exposure to PCBs may be identified. The first group includes those persons with the potential for frequent or high exposure, namely, occupationally-exposed workers and breast-fed infants, as PCBs are excreted in the breast milk of lactating humans (Miller, 1977; Rogan et al., 1980; Wickizer et al., 1981; Mes and Davies, 1979; Kuwabara et al., 1979; Hofvander et al., 1981). The second group includes those persons with an inability to oxidize PCBs via glucuronidation to facilitate detoxification and elimination of these toxicants, such as embryos, fetuses, and neonates (2 to 3 months old) (Calabrese and Sorenson, 1977; Gillette, 1967; Nyhan, 1961), especially breast-fed infants who receive a steroid via human breast milk that inhibits glucuronyl transferase activity (Calabrese and Sorenson, 1977; Gartner and Arias, 1966), children simultaneously exposed to the antibiotic novobiocin (Lokietz et al., 1963; Calabrese and Sorenson, 1977), persons with Gilbert's syndrome or Crigler and Najjar syndrome (Lester and Schmid, 1964; Calabrese and Sorenson, 1977), or persons with hepatic infections such as infectious hepatitis (Calabrese and Sorenson, 1977).

COCARCINOGENESIS, INITIATION, AND PROMOTION

DiGiovanni et al. (1978) demonstrated that Aroclor 1254 had weak tumor initiating activity in the mouse two-stage tumorigenesis models. Promoting activity was not indicated for this Aroclor in a study by Berry et al. (1978). Using other experimental systems, Ito et al. (1973) observed an increased incidence of liver tumors in rats co-administered PCBs and benzene hexachloride as compared with benzene hexachloride treatment alone. Co-administration of other potent live carcinogens, 3'-methyl-4-dimethylaminoazobenzene, N-2-fluorenylacetemide and diethylnitrosamine, with PCBs, however, has resulted in the inhibition of the tumorigenic response (Ito et al., 1973). Similarly, antineoplastic effects were reported by Nishizumi (1980) for pups of dams administered Kanechlor-500 and diethylnitrosamine. These studies make it apparent that exposure to PCBs can affect the carcinogenicity of other xenobiotics.

SUMMARY

For the purposes of setting advisory levels for PCBs contaminating soils, acceptable intake (AI) levels have been developed which are based on both the toxicity and the carcinogenicity of PCBs. The 10-day health advisories (HA) for toxicity other than cancer have been developed. The 1-day and lifetime HAs could not be evaluated. Advisories for the cancer end point are expressed in terms of 10^{-4} to 10^{-7} lifetime individual excess risk levels. See Table D-3 for a summary of these AIs and risk-specific doses.

TABLE D-3. SUMMARY OF RISK SPECIFIC DOSES (RSDs) FOR CANCER RISKS OF PCBs, OR OF ACCEPTABLE INTAKES FOR PROTECTION AGAINST THE NONCARCINOGENIC EFFECTS OF PCBs

Description	Value (μg/day)
10^{-4} RSD	1.75
10^{-5} RSD	0.175
10^{-6} RSD	0.0175
Short-term AI (1-day)	Not estimated
Longer-term AI (10-day)	100 for a child
	700 for an adult
Lifetime AI	Not estimated

The toxicity and carcinogenicity of PCBs have recently been critically evaluated in a number of EPA documents (U.S. EPA, 1980, 1983, 1984b, 1985b). The approach taken here in developing a 10-day exposure advisory for non-cancer toxicity has been to select from the available literature the animal study that addresses the critical toxic effect and yields the most appropriate no observed effect, no observed adverse effect, lowest observed effect, or lowest observed adverse effect level. This dose was then divided by an appropriate uncertainty factor to obtain the 10-day HA.

The calculation of a 10-day HA for noncarcinogenic toxicity should make use of animal data derived after an exposure period ranging from 10 to 30 days. The literature contains several animal studies which address this length of exposure. The studies chosen as a basis for the 10-day HA (Villeneuve et al., 1971; Grant and Phillips, 1974; Collins and Capen, 1980a, b, c; Carter, 1983) yield a No Observed Adverse Effect Level (NOAEL). These studies involved the feeding of Aroclor 1254 to rabbits and rats. Collectively, in these studies, a NOAEL can be ascribed to a dose of 1 mg/kg/day). Dividing this NOAEL by an uncertainty factor of 100 yields a 10-day HA of 100 μg/kg/day for a child, and 700 μg/day for a 70-kg adult.

The 1-day HA could not be estimated based on animal data derived from studies with an exposure duration of 1 to 5 days. The lifetime HA noncancer toxicity, likewise, could not be estimated.

The cancer risk specific doses (intake levels) have been calculated by solving for exposure in the equation

$$\text{Risk} = \text{Potency} \times \text{Exposure}$$

and multiplying by 70 kg. A cancer potency factor of 4.0 $(\text{mg/kg/day})^{-1}$ was adopted for use in these calculations. This value is the mean of the values determined by OTS (3.57 $(mg/kg/day)^{-1}$) and by ORD (4.34 $(mg/kg/day)^{-1}$) for PCBs.

REFERENCES (APPENDIX D)

Allen, J.R. (1975) Response of the non-human primate to polychlorinated biphenyls exposure. Fed. Proc. 34:1675-1679.

Allen, J.R.; Abrahamson, L.J. (1979) Responses of rats exposed to polychlorinated biphenyls for 52 weeks. II. Compositional and enzymic changes in the liver. Arch. Environ. Contam. Toxicol. 8:191.

Allen, J.R.; Barsotti, D.A. (1976) The effects of transplacental and mammary movement of the PCBs on infant rhesus monkeys. Toxicology 6:331-340.

Allen, J.R.; Barsotti, D.A.; Carstens, L.A. (1980) Residual effects of polychlorinated biphenyls on adult nonhuman primates and their offspring. J. Toxicol. Environ. Health 6(1):55-66.

Allen, J.R.; Carsten, L.A.; Abrahamson, L.J. (1976) Responses of rats exposed to polychlorinated biphenyls for 52 weeks. I. Comparison of tissue levels of PCB and biological changes. Arch. Environ. Contam. Toxicol. 4:409.

Allen, J.R.; Norback, D.H.; Hsu, I.C. (1974) Tissue modifications in monkeys as related to absorption, distribution, and excretion of polychlorinated biphenyls. Arch. Environ. Contam. Toxicol. 2:86.

American Conference of Governmental Industrial Hygienists (ACGIH). (1980) Documentation of the threshold limit values. 4th ed. (Includes supplemental documentation, 1981). Cincinnati, OH, p. 486.

Aulerich, R.J.; Bursian, S.J.; Breslin, W.J.; Olson, B.A.; Ringer, R.K. (1985) Toxicological manifestations of 2,4,5-2',4',5'-, 2,3,6,2',3',6'-, and 3,4,5,3',4',5'-hexachlorobiphenyl and Aroclor 1254 in mink. J. Toxicol. Environ. Health 15:63-79.

Aulerich, R.J.; Ringer, R.K. (1977) Current status of PCB toxicity to mink, and effect on their reproduction. Arch. Environ. Contam. Toxicol. 6:279-292.

Bahn, A.K.; Grover, P.; Rosenwaike, I.; O'Leary, K.; Stellman, J. (1977) PCBs and melanoma. N. Gig. J. Med. 296:108.

Bahn, A.K.; Rosenwaike, I.; Herrmann, N.; Grover, P.; Stellman, J.; O'Leary, K. (1976) Melanoma after exposure to PCBs. New Engl. J. Med. 295:450.

Baker, F.D.; Bush, B.; Tumasonis, C.F.; Lo, F.C. (1977) Toxicity and persistence of low-level PCB in adult Wistar rats, fetuses, and young. Arch. Environ. Contam. Toxicol. 5(2):143-156.

Barsotti, D.A.; Allen, J.R. (1975) Effects of polychlorinated biphenyls on reproduction in the primate. Fed. Proc. 34:338.

Becker, G.M.; McNulty, W.P.; Bell, M. (1979) Polychlorinated biphenyls-induced morphologic changes in the gastric mucosa of the rhesus monkey. Lab. Invest. 40:373.

Bell, M. (1983) Ultrastructural features of the murine cutaneous microvasculature after exposure to polychlorinated biphenyl compounds (PCBs) and benzo(a)pyrene (BAP). Virchows Arch. B. 42(2):131-142.

Berry, D.L.; Giovanni, J.D.; Juchau, M.R.; Bracken, W.M.; Gleason, G.L.; Slaga, T.J. (1978) Lack of tumor-promoting ability of certain environmental chemicals in a two-stage mouse skin tumorigenesis assay. Res. Commun. Chem. Pathol. Pharmacol. 20:101.

Biocca, M.; Gupta, B.N.; Chae, K.; McKinney, J.D.; Moore, J.A. (1981) Toxicity of selected symmetrical hexachlorobiphenyl isomers in the mouse. Toxicol. Appl. Pharmacol. 58:461-474.

Bleavins, M.R.; Aulerich, R.J.; Ringer, R.K. (1980) Polychlorinated biphenyls (Aroclors 1016 and 1242): effects on survival and reproduction in mink and ferrets. Arch. Environ. Contam. Toxicol. 9(5):627-635.

Bruckner, J.V.; Khanna, K.L.; Cornish, H.H. (1973) Biological responses of the rat to polychlorinated biphenyls. Toxicol. Appl. Pharmacol. 24:434-448.

Brunstroem, B.; Kihlstroem, I.; Lundkvist, U. (1982) Studies of fetal death and fetal weight in guinea pigs fed polychlorinated biphenyls (PCBs). Acta. Pharmacol. Toxicol. 50(2):100-103.

Calabrese, E.J.; Sorenson, A.J. (1977) The health effects of PCBs with particular emphasis on human high-risk groups. Rev. Environ. Health 2(4): 285-304.

Carter, J.W. (1983) Onset of hepatomegaly in PCB (Aroclor 1254)-treated rats. Bull. Environ. Contam. Toxicol. 31(2):183-187.

Carter, J.W.; Mercer, L.P. (1983) Prediction of food intake, weight gain, and organ weights in rats as a formulation of dietary Aroclor 1254 (PCBs) by the saturation kinetics model for physiological responses. Nutr. Rep. Int. 27:561-568.

Collins, W.T.; Capen, C.C. (1980a) Biliary excretion of thyroixine-I-125 and fine structural alterations in the thyroid glands of gunn-rats fed PCBs. Lab. Invest. 43:158.

Collins, W.T.; Capen, C.C. (1980b) Ultrastructural and functional alterations of the rat thyroid gland produced by polychlorinated biphenyls compared with iodide excess and deficiency, and thyrotropin and thyroxine administration. Virchows Arch. B. 33(3):213-231.

Collins, W.T.; Capen, C.C. (1980b) Fine structural lesions and hormonal alterations in thyroid glands of perinatal rats exposed in utero and by the milk to polychlorinated biphenyls. Am. J. Pathol. 99(1):125-141.

Davidorf, F.H.; Knupp, J.A. (1979) Epidemiology of ocular melanoma. Incidence and geographic relationship in Ohio (1967-1977). Ohio State Med. J. 75(9):561-564.

DiGiovanni, J.; Slaga, T.J.; Berry, D.L.; Juchan, M.R. (1978) The effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and Aroclor 1254 on 7,12-dimethylbenz[A]anthracene metabolism and tumor-initiating activity in mouse skin. Proc. AACR, ASCO 19:110.

Dikshith, T.S.S.; Rockwood, W.; Abraham, R.; Coulston, F. (1975) Effects of polychlorinated biphenyls (Aroclor 1254) on rat testis. Exp. Mol. Pathol. 22:376.

Fishbein, L. (1974) Toxicity of chlorinated biphenyls. Ann. Rev. Pharmacol. 14:139.

Garthoff, L.H.; Cerra, F.E.; Marks, E.M. (1981) Blood chemistry alterations in rats after single and multiple gavage administration of polychlorinated biphenyls. Toxicol. Appl. Pharmacol. 60(1):33-44.

Garthoff, L.H.; Friedman, L.; Farber, T.M.; Locke, K.K.; Sobotka, T.J.; Green, S.; Hurley, N.E.; Peters, E.L.; Story, G.E.; Moreland, F.M.; Graham, C.H.; Keys, J.E.; Taylor, M.J.; Scalera, J.V.; Rothlein, J.E.; Marks, E.M.; Cerra, F.E.; Rodi, S.B.; Sporn, E.M. (1977) Biochemical and cytogenetic effects in rats caused by short-term ingestion of Aroclor 1254 or Firemaster BP6. J. Toxicol. Environ. Health 3:769-796.

Gartner, L.M.; Arias, I.M. (1966) Studies of prolonged neonatal jaundice in the breast-fed infant. J. Pediat. 68(1):54.

Gillette, J.R. (1967) Individually different responses to drugs according to age, sex, and functional or pathological state. In: Wolstenholme, G.; Porter, R., eds. Drug responses to man. Churchill, London, p. 28.

Grant, D.L.; Phillips, W.E.J. (1974) The effect of age and sex on the toxicity of Aroclor 1254, a polychlorinated biphenyl, in the rat. Bull. Environ. Contam. Toxicol. 12:145-152.

Green, S.; Sauro, F.M.; Friedman, L. (1975) Lack of dominant lethality in rats treated with polychlorinated biphenyls (Aroclors 1242 and 1254). Food Cosmet. Toxicol. 13:507-510.

Haque, R.; Schmedding, D.W.; Freed, V.H. (1974) Aqueous solubility, adsorption and vapor behavior of polychlorinated biphenyl Aroclor 1254. Environ. Sci. Technol. 8(2):139-142.

Haque, R.; Schmedding, D.W. (1975) A method of measuring the water solubility of hydrophobic chemicals: solubility of five polychlorinated biphenyls. Bull. Environ. Contam. and Toxicol. 14(1):13-18.

Heddle, J.A.; Bruce, W.R. (1977) Comparison of tests for mutagenicity or carcinogenicity using assays for sperm abnormalities, formation of micronuclei, and mutations in Salmonella. In: Hiatt, H.H., ed. Origins of human cancer: proceedings of conference on cell proliferation, Cold Spring Harbor, NY. Vol. 4, p. 1549.

Hofvander, Y.; Hagman, U.; Linder, C.E.; Vaz, R.; Slorach, S.A. (1981) World Health Organization collaborative breast feeding study. I. Organochlorine contaminants in individual samples of Swedish human milk, 1978-1979. Acta. Paediatr. Scand. 70(1):3-8.

International Agency for Research on Cancer (IARC). (1978) Polychlorinated biphenyls. In: IARC monographs on the evaluation of the carcinogenesis risk of chemicals to humans, Vol. 18, p. 43. Lyon, France.

International Agency for Research on Cancer (IARC). (1982) IARC monographs on the evaluation of the carcinogenesis risk of chemicals to humans. Supplement 4. Lyon, France.

Ito, N.; Nagasaki, H.; Arai, M.; Makiura, S.; Sugihara, S.; Hirao, K. (1973) Histopathologic studies on liver tumorigenesis induced in mice by technical polychlorinated biphenyls and its promoting effect on liver tumors induced by benzene hexachloride. J. Natl. Cancer Inst. 51:1637-1646.

Ito, N.; Nagasaki, Makiura, S.; H.; Arai, M. (1974) Histopathological studies on liver tumorigenesis in rats treated with polychlorinated biphenyls. Gann. 65:545-549.

Jonsson, H.T. Jr.; Walker, E.M.; Green, W.B.; Hughson, M.D.; Hennigar, G.R. (1981) Effeft of prolonged exposure to dietary DDT and PCB on rat liver morphology. Arch. Environ. Contam. Toxicol. 10(2):171-183.

Kasza, L.; Collins, W.T.; Capen, C.C.; Garthoff, L.H.; Friedman, L. (1978a) Comparative toxicity of polychlorinated biphenyls and polybrominated biphenyl in the rat thyroid gland: light and electron microscope alterations after subacute dietary exposure. J. Environ. Pathol. Toxicol. 1:587-599.

Kasza, L.; Weinberger, M.A.; Hinton, D.E. (1978b) Comparative toxicity of polychlorinated biphenyls and polybrominated biphenyl in the rat thyroid gland: light and electron microscope alterations after subacute dietary exposure. J. Environ. Pathol. Toxicol. 1:241-257.

Kimbrough, R.; Buckley, J.; Fishbein, L.; Flamm, G.; Kasza, L.; Marcus, W.; Shibko, S.; Teske, R. (1978) Animal toxicity. Environ. Health Perspect. 24:173-184.

Kimbrough, R.D.; Linder, R.E. (1974) Induction of adenofibrosis and hepatomas in the liver of Balb/cJ mice by polychlorinated biphenyls (Aroclor 1254). J. Natl. Cancer Inst. 53:547-579.

Kimbrough, R.D.; Linder, R.E.; Gaines, T.B. (1972) Morphological changes in livers of rats fed polychlorinated biphenyls. Arch. Environ. Health 25:354.

Kimbrough, R.D.; Squire, R.A.; Linder, R.E.; Strandberg, J.D.; Montali, R.J.; Burse, V.W. (1975) Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyls (Aroclor 1260). J. Natl. Cancer Inst. 55:1453-1459.

Kimura, N.T.; Baba, T. (1973) Neoplastic changes in the rat liver induced by polychlorinated biphenyls. Gann. 64:105-108.

Koller, L.D. (1977) Enhanced polychlorinated biphenyls lesions in Moloney leukemia virus-infected mice. Clin. Toxicol. 11(1):107-116.

Komives, G.K. (1979) Body weights, food intakes, and water intakes in rats during daily administration of closely controlled doses of polychlorinated biphenyls. Bull. Environ. Contam. Toxicol. 22(6):761-766.

Komives, G.K.; Alayoku, G. (1980) Body temperatures and weights in rats during daily administration of closely controlled doses of polychlorinated biphenyls. Bull. Environ. Contam. Toxicol. 25(6):913-917.

Kuratsune, M. (1976) Epidemiologic studies on Yusho. In: Higuchi, K., ed. PCB poisoning and pollution. pp. 9-23.

Kuwabara, K.; Yakushiji, T.; Wathanabe, I.; Yoshida, S.; Kyoama, K.; Kunita, N. (1979) Levels of polychlorinated biphenyls in blood of breast-fed children whose mothers are non-occupationally exposed to PCBs. Bull. Environ. Contam. Toxicol. 21(4-5):458-462.

Lester, R.; Schmid, R. (1964) Bilirubin metabolism. New Engl. J. Med. 270(15):779.

Lewin, C.; McBlain, W.A.; Wolfe, F.H. (1972) Acute intraperitoneal toxicity of DDT and PCBs in mice using two solvents. Bull. Environ. Contam. Toxicol. 8(4):245-250.

Linder, R.E.; Gaines, T.B.; Kimbrough, R.D. (1974) The effect of PCB on rat reproduction. Food Cosmet. Toxicol. 12:63-77.

Lokietz, H.; Dowben, R.M.; Hsia, D.Y. (1963) Studies on the effect of novobiocin and glucuronyl transferase. Pediatrics 32:47.

Loose, L.D.; Pittman, K.A.; Benitz, K.-F.; Sitknorth, J.B.; Mueller, W.; and Coulston, F. (1978) Environmental chemical-induced immune dysfunction. Ecotoxicol. Environ. Safety 2:173.

Mes, J.; Davies, D.J. (1979) Presence of polychlorinated biphenyls and organochlorine pesticide residues and the absence of polychlorinated terphenyls in Canadian human milk samples. Bull. Envion. Contam. Toxicol. 21(3):381-387.

McConnell, E.E.; Kinney, J.D. (1978) Exquisite toxicity in the guinea pig to structurally similar halogenated dioxins, furans, biphenyls, and naphthalenes. Toxicol. Appl. Pharmacol. 45:298.

Mieure, J.P.; Hicks, O.; Kaley, R.G.; Saeger, V.W. (1976) Characterization of polychlorinated biphenyls. National Conference on Polychlorinated Biphenyls (Nov. 19-21, 1975, Chicago, IL). EPA-560/6-75-004. U.S. Environmental Protection Agency, Washington, D.C.

Miller, J.W. (1944) Pathologic changes in animals exposed to a commercial chlorinated diphenyl. Pub. Health Rep. 59:1085.

Miller, R.W. (1977) Pollutants in breast milk: PCBs and cola-colored babies (editorial). J. Pediatr. 90(3):510-511.

Morgan, R.W.; Ward, J.M.; Hartman, P.E. (1981) Aroclor 1254-induced intestinal metaplasia and adenocarcinoma in the glandular stomach of F344 rats. Cancer Res. 41(12)5052-5059.

Narbonne, J.F. (1979) Effects of age and sex on liver responses of Phenoclor DP6, a polychlorinated biphenyl, in the rat. Bull. Environ. Contam. Toxicol. 22(1):49-54.

National Academy of Sciences (NAS). (1980) Drinking water and health, Vol. 3. Washington, D.C.: National Academy Press, p. 133.

National Cancer Institute (NCI). (1978) Bioassay of Aroclor (trademark) 1254 for possible carcinogenicity. CAS No. 27323-18-8. Carcinogenesis Technical Report Series No. 38. Bethesda, MD.

National Institute for Occupational Safety and Health (NIOSH). (1977) Criteria for a recommended standard...occupational exposure to polychlorinated biphenyls (PCBs). U.S. Department of Health, Education, and Welfare.

Nelson, N.; Hammon, B.; Nisbet, C.T.; Sarafirm, A.F.; Dewey, W.H. (1972) Polychlorinated biphenyls: environmental impact. Environ. Res. 5:249.

Nilsson, B.; Ramel, C. (1974) Genetic tests on Drosophila melanogaster with polychlorinated biphenyls (PCBs). Hereditas 77:319-322.

Nisbet, I.C.T.; Sarofim, A.F. (1972) Rates and routes of transport of PCBs in the environment. Environ. Health Perspect. 1:21.

Nishizumi, M. (1980) Reduction of diethylnitrosamine-induced hepatoma in rats exposed to polychlorinated biphenyls through their dams. Gann 71(6):910-912.

Nyhan, W.L. (1961) Toxicity of drugs in the neonatal period. J. Pediat. 59(1):1.

Orberg, J.; Kihlstrom, J.E. (1973) Effects of long-term feeding of polychlorinated biphenyls (PCB, Clophen A60) on the length of the oestrous cycle and on the frequency of implanted ova in the mouse. Environ. Res. 6:176.

Peakall, D.B.; Lincer, J.L.; Bloom, S.E. (1972) Embryonic mortality and chromosomal alterations caused by Aroclor 1254 in ring doves. Environ. Health Perspect. 1:103-104.

Rogan, W.J.; Bogniewska, A.; Damstra, T. (1980) Pollutants in breast milk. New Engl. J. Med. 302:1450.

Rosin, D.L.; Martin, B.R. (1983) Comparison of the effects of acute and subchronic administration of Aroclor 1254, a commercial mixture of polychlorinated biphenyls, on pentobarbital-induced sleep time and [C]pentobarbital disposition in mice. J. Toxicol. Environ. Health 11:917-931.

Sager, D.B. (1983) Effect of postnatal exposure to polychlorinated biphenyls on adult male reproduction function. Environ. Res. 31(1):76-94.

Sanders, O.T.; Zepp, R.L.; Kirkpatrick, R.L. (1974) Effect of PCB ingestion on sleeping times, organ weights, food consumption, serum corticosterone and survival of albino mice. Bull. Environ. Contam. Toxicol. 12(4):394-399.

Schoeny, R.S. (1982) Mutagenicity testing of chlorinated biphenyls and chlorinated dibenzofurans. Mutat. Res. 101:45.

Schoeny, R.S.; Smith, C.C.; Loper, J.C. (1979) Non-mutagenicity for Salmonella of the chlorinated hydrocarbons Aroclor 1254, 1,2,4-trichlorobenzene, mirex, and kepone. Mutat. Res. 68:125-132.

Spencer, F. (1982) An assessment of the reproductive toxic potential of Aroclor 1254 in female Sprague-Dawley rats. Bull. Environ. Contam. Toxicol. 28(3):290-297.

Street, J.C.; Sharma, R.P. (1975) Alteration of induced cellular and humoral immune responses by pesticides and chemicals of environmental concern: quantitative studies of immunosuppression by DDT, Aroclor 1254, carbaryl, carbofuran, and methyl parathion. Toxicol. Appl. Pharmacol. 32:587.

Urabe, H. (1974) Foreword to the fourth report of the Study Group on Yusho and PCB. Fukuoka Acta. Med. 65:145. (In Japanese).

U.S. Environmental Protection Agency. (1980) Ambient water quality criteria document for PCBs. Environmental Criteria and Assessment Office, Cincinnati, OH. EPA-440/5-80-068. Available from: National Technical Information Service, Springfield, VA; PB81-117798.

U.S. Environmental Protection Agency. (1983) Quantitative risk assessment of reproductive risks associated with polychlorinated biphenyl (PCB) exposure. Support Document B, Docket No. OPTS-62032 PCB.

U.S. Environmental Protection Agency. (1984a) Proposed guidelines for carcinogen risk assessment; request for comments. Federal Register 49(227): 46294-46301.

U.S. Environmental Protection Agency. (1984b) Health effects assessment for polychlorinated biphenyls. Environmental Criteria and Assessment Office, Cincinnati, OH. Final draft. EPA-540/1-86/004. Available from: National Technical Information Service, Springfield, VA; PB86-134152/AS.

U.S. Environmental Protection Agency. (1985a, June 12) Personal communication, Don R. Clay, Director, Office of Toxic Substances.

U.S. Environmental Protection Agency. (1985b) Drinking water criteria document for polychlorinated biphenyls (PCBs). Environmental Criteria and Assessment Office, Cincinnati, OH. Final draft. EPA-600/X-84-198. Available from: National Technical Information Service, Springfield, VA; PB118312/AS.

Villeneuve, D.C.; Grant, B.L.; Khera, K.; Clegg, D.J.; Baer, H.; and Phillips, W.E.J. (1971) The fetotoxicity of a polychlorinated biphenyl mixture (Aroclor 1254) in the rabbit and in the rat. Environ. Physiol. 1:67-71.

Vos, J.G.; van Genderen, H. (1973) Toxicological aspects of immunosuppression. In: Deichman, W.B., ed. Pesticides in the environment: a continuing controversy; 8th international conference on toxicology and occupational medicine, Miami, FL. New York, NY: Intercontinental Medical Book Co.

Wickizer, T.M.; Brilliant, L.B.; Copeland, R.; Tilden, R. (1981) Polychlorinated biphenyl contamination of nursing mothers' milk in Michigan (USA). Am. J. Public Health 71(2):132-137.

Wyndham, C.; Devenish, J.; Safe, S. (1976) The in vitro metabolism, macromolecular binding, and bacterial mutagenicity of 4-chlorobiphenyl, a model PCB substrate. Res. Commun. Chem. Pathol. Pharmacol. 15:563.

Zinkl, J.G. (1977) Skin and liver lesions in rats fed a polychlorinated biphenyl mixture. Arch. Environ. Contam. Toxicol. 5(2):217-228.